```
     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 21:03:33 ON 23 SEP 2002
L1           634 S (CALCIUM OR STRONTIUM OR CANO3 OR CACL2) (50A) (ALLERGEN? OR
L2         14418 S (DENATUR? OR CONTROL? OR ELIMINAT? OR REDUC? OR DEACTIVAT? OR
L3           153 S L2 AND L1
L4           139 DUP REM L3 (14 DUPLICATES REMOVED)
L5           574 S (CALCIUM OR STRONTIUM OR CANO3 OR CACL2) (25A) (ALLERGEN? OR
L6           117 S L5 AND L4
L7            43 S L6 AND ALLERG?
L8            59 S L4 AND ALLERG?

=> d que l8
L1           634 SEA (CALCIUM OR STRONTIUM OR CANO3 OR CACL2) (50A) (ALLERGEN?
                 OR POLLEN# OR DUSTMITE# OR MITE#)
L2         14418 SEA (DENATUR? OR CONTROL? OR ELIMINAT? OR REDUC? OR DEACTIVAT?
                 OR (DE (W) ACTIVAT?) OR INACTIVAT?) (50A) (ALLERGEN? OR
                 POLLEN# OR DUSTMITE# OR MITE#)
L3           153 SEA L2 AND L1
L4           139 DUP REM L3 (14 DUPLICATES REMOVED)
L8            59 SEA L4 AND ALLERG?
```

(L9) L4 not L8

Reviewed online

(L14) — Reviewed mem — All no good

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     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 21:03:33 ON 23 SEP 2002
L1          634 S (CALCIUM OR STRONTIUM OR CANO3 OR CACL2) (50A) (ALLERGEN? OR
L2        14418 S (DENATUR? OR CONTROL? OR ELIMINAT? OR REDUC? OR DEACTIVAT? OR
L3          153 S L2 AND L1
L4          139 DUP REM L3 (14 DUPLICATES REMOVED)
L5          574 S (CALCIUM OR STRONTIUM OR CANO3 OR CACL2) (25A) (ALLERGEN? OR
L6          117 S L5 AND L4
L7           43 S L6 AND ALLERG?
L8           59 S L4 AND ALLERG?
L9           80 S L4 NOT L8

     FILE 'STNGUIDE' ENTERED AT 21:15:04 ON 23 SEP 2002
L10           0 S (ALLERG?) (10A) NEUTRALIZ?

     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 21:46:18 ON 23 SEP 2002
L11         168 S (ALLERG?) (10A) NEUTRALIZ?
L12          10 S (L11 AND (CALCIUM OR STRONTIUM)) NOT CALCIUM CHANNEL#
L13           8 DUP REM L12 (2 DUPLICATES REMOVED)
L14           4 S L13 NOT L8

     FILE 'REGISTRY' ENTERED AT 21:49:02 ON 23 SEP 2002
L15           1 S STRONTIUM CHLORIDE/CN

     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 21:49:22 ON 23 SEP 2002

     FILE 'REGISTRY' ENTERED AT 21:49:40 ON 23 SEP 2002
               SET SMARTSELECT ON
L16          SEL L15 1- CHEM :        4 TERMS
               SET SMARTSELECT OFF

     FILE 'CAPLUS, WPIDS, MEDLINE' ENTERED AT 21:49:41 ON 23 SEP 2002
L17        4229 S L16/BI
L18          20 S (L17 OR STRONTIUM) (L) (ALLERG? OR DUSTMITE# OR MITE#)
L19          19 DUP REM L18 (1 DUPLICATE REMOVED)
L20          16 S L19 NOT (L4 OR L14)

=> d que 18
L1          634 SEA (CALCIUM OR STRONTIUM OR CANO3 OR CACL2) (50A) (ALLERGEN?
               OR POLLEN# OR DUSTMITE# OR MITE#)
L2        14418 SEA (DENATUR? OR CONTROL? OR ELIMINAT? OR REDUC? OR DEACTIVAT?
               OR (DE (W) ACTIVAT?) OR INACTIVAT?) (50A) (ALLERGEN? OR
               POLLEN# OR DUSTMITE# OR MITE#)
L3          153 SEA L2 AND L1
L4          139 DUP REM L3 (14 DUPLICATES REMOVED)
L8           59 SEA L4 AND ALLERG?
```

Fully Printed out

Reviewed online. Vast majority were bad hits. Relevant hits printed out individually.

Reviewed online. All no good

Strontium Focus

Fully Printed out

L8 ANSWER 1 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 2002:275734 CAPLUS
DN 136:305488
TI **Allergen** neutralization compositions
IN Hasan, Abul Khaer Mohamad Quamrul; Mao, Mark Hsiang-Kuen; Kobayashi, Ryoko
PA The Procter & Gamble Company, USA
SO PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002028187	A1	20020411	WO 2000-US27019	20000929

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI **Allergen** neutralization compositions
AB **Allergen** neutralization compns. for use on inanimate objects having an effective amt. of a **allergy** neutralizing metal ion, and a solvent. The **allergen** neutralization compns. are sprayable, and preferably contain **allergen denaturing** compds. such as polyphenol compds., hydrogen peroxide, salicylic acid, citric acid, lactic acid, glycolic acid, and mixts. of these. The metal ion is selected from ions of zinc, stannous, stannic, magnesium, calcium, manganese, titanium, iron, copper, nickel, and mixts. of these. These **allergen** neutralization compns. provide excellent efficacy against various **allergens**, and specifically, the **allergens** assocd. with house dust mites and other common **allergens** such as cat dander, pollen and the like. Moreover, these compns. do not stain common household surfaces.
ST metal ion sprayable **allergen** neutralization compn; nickel sprayable **allergen** neutralization compn; copper sprayable **allergen** neutralization compn; iron sprayable **allergen** neutralization compn; titanium sprayable **allergen** neutralization compn; manganese sprayable **allergen** neutralization compn; **calcium** sprayable **allergen** neutralization compn; magnesium sprayable **allergen** neutralization compn; tin sprayable **allergen** neutralization compn; zinc sprayable **allergen** neutralization compn; glycolic acid sprayable **allergen** neutralization compn; lactic acid sprayable **allergen** neutralization compn; citric acid sprayable **allergen** neutralization compn; salicylic acid sprayable **allergen** neutralization compn; hydrogen peroxide sprayable **allergen** neutralization compn; polyphenol compd sprayable **allergen** neutralization compn
IT Phenols, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(polyphenols, nonpolymeric; sprayable **allergen** neutralization compns.)
IT Dermatophagoides
Pollen

(sprayable **allergen** neutralization compns.)

IT **Allergens**
RL: BCP (Biochemical process); REM (Removal or disposal); BIOL (Biological study); PROC (Process)
(sprayable **allergen** neutralization compns.)

IT Tannins
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(sprayable **allergen** neutralization compns.)

IT 50-21-5, Lactic acid, biological studies 69-72-7, Salicylic acid, biological studies 77-92-9, Citric acid, biological studies 79-14-1, Glycolic acid, biological studies 7439-89-6, Iron, biological studies 7439-95-4, Magnesium, biological studies 7439-96-5, Manganese, biological studies 7440-02-0, Nickel, biological studies 7440-31-5, Tin, biological studies 7440-32-6, Titanium, biological studies 7440-50-8, Copper, biological studies 7440-66-6, Zinc, biological studies 7440-70-2, **Calcium**, biological studies 7488-55-3, Stannous sulfate 7646-85-7, Zinc chloride, biological studies 7720-78-7, Ferrous sulfate 7722-84-1, Hydrogen peroxide, biological studies 7758-94-3, Ferrous chloride 7772-99-8, Stannous chloride, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(sprayable **allergen** neutralization compns.)

L8 ANSWER 2 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 2002:275727 CAPLUS
DN 136:290411
TI **Allergen** neutralization compositions
IN Hasan, Abul Khaer Mohamad Quamrul; Mao, Mark Hsiang-Kuen; Kobayashi, Ryoko
PA The Procter & Gamble Company, USA
SO PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002028179	A1	20020411	WO 2000-US27018	20000929

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI **Allergen** neutralization compositions
AB **Allergen** neutralization compns. that retain at least about 30% of dust particles as measured by the Dust **Control** Test, and the compns. have an av. MIU value of less than 3.4 as measured by the Friction Coeff. Anal. method. The compns. preferably contain a film forming polymer to control dust while maintaining a smooth feeling on the surface being treated. These **allergen** neutralization compns. are for use on inanimate objects, and are sprayable. Preferably these **allergen** neutralization compns. contain **allergen** **denaturing** compds. such as an effective amt. of an **allergy** neutralizing metal ion, polyphenol compds., hydrogen peroxide, salicylic acid, citric acid, lactic acid, glycolic acid, and mixts. of theses. By

controlling dust particles that contain **allergenic** proteins, these **allergen** neutralization compns. provide excellent efficacy against various **allergens**, and specifically, the **allergens** assocd. with house dust **mites** and other common **allergens** such as cat dander, **pollen** and the like.

ST **allergen** neutralization compn; dust particle

IT Dermatophagoides
Dust
Pollen
(**allergen** neutralization compns.)

IT **Allergens**
RL: BCP (Biochemical process); REM (Removal or disposal); BIOL (Biological study); PROC (Process)
(**allergen** neutralization compns.)

IT Polyoxyalkylenes, biological studies
Trace metals
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(**allergen** neutralization compns.)

IT Dust
(house; **allergen** neutralization compns.)

IT Phenols, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(polyphenols, nonpolymeric; **allergen** neutralization compns.)

IT 50-21-5, Lactic acid, biological studies 50-81-7, Ascorbic acid, biological studies 69-72-7, Salicylic acid, biological studies 77-92-9, Citric acid, biological studies 79-14-1, Glycolic acid, biological studies 111-46-6, Diethylene glycol, biological studies 149-91-7, Gallic acid, biological studies 526-95-4, Gluconic acid 7439-89-6, Iron, biological studies 7439-95-4, Magnesium, biological studies 7439-96-5, Manganese, biological studies 7440-02-0, Nickel, biological studies 7440-32-6, Titanium, biological studies 7440-50-8, Copper, biological studies 7440-66-6, Zinc, biological studies 7440-70-2, **Calcium**, biological studies 7488-55-3, Stannous sulfate 7646-85-7, Zinc chloride, biological studies 7720-78-7, Ferrous sulfate 7722-84-1, Hydrogen peroxide, biological studies 7758-94-3, Ferrous chloride 7772-99-8, Stannous chloride, biological studies 9002-89-5, Polyvinyl alcohol 9003-01-4, Polyacrylic acid 9003-39-8, Poly(vinylpyrrolidone) 9004-67-5, Methyl cellulose 10476-85-4, **Strontium** chloride 25322-68-3, Polyethylene glycol 25322-69-4, Polypropylene glycol 26062-79-3, Polyquaternium 6
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(**allergen** neutralization compns.)

L8 ANSWER 3 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 2001:879743 CAPLUS
DN 136:16288
TI Effects of the inhalation of diesel exhaust, kanto loam dust, or diesel exhaust without particles on immune responses in mice exposed to Japanese cedar (Cryptomeria Japonica) pollen
AU Maejima, Kazuhito; Tamura, Kumiko; Nakajima, Toru; Taniguchi, Yoshifumi; Saito, Saburo; Takenaka, Hiroshi
CS Japan Automobile Research Institute, Ibaraki, 305-0822, Japan
SO Inhalation Toxicology (2001), 13(11), 1047-1063
CODEN: INHTE5; ISSN: 0895-8378
PB Taylor & Francis
DT Journal
LA English
RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB To assess the potential enhancement by air-pollutants of immune responses in mice, esp. with regard to **allergen**-specific IgE antibody prodn., female BDF1 mice (60 mice in each group) were exposed to diesel exhaust (particles, 3.24 mg/m3; nitrogen dioxide, 1.0 ppm: DE group), Kanto loam dust (particles, 3.29 mg/m3; nitrogen dioxide 0.01 ppm: KLD group), diesel exhaust without particles (particles, 0.01 mg/m3; nitrogen dioxide, 1.1 ppm: DEG group), or clean air (**pollen** and **control** groups) for 16 h/day, 5 days/wk for 24 wk, as well as to Japanese cedar **pollen** (JCP) (around 550,000 grains of JCP/m3) for 2 days/wk in the same period. The control group was exposed to clean air alone throughout the expt. The mean values for Japanese cedar pollen **allergens** (JCPAs)-specific IgE antibody titers in mice sera measured by ELISA in the DE, KLD, and DEG groups were higher than that for the pollen alone group, but not significantly, after both 12 and 24 wk of exposure time. The percentages of animals expressing more than the min. ELISA titer of JCPAs-specific IgE antibodies in each group were 22% (DE and pollen groups) and 27% (KLD and DEG groups) of the totals at wk 12, and no statistical differences were obsd. among the groups. However, at wk 24 in the DE, KLD, and DEG groups the responders comprised 73%, 63%, and 67%, resp., significantly higher than the 33% for the pollen alone group. No significant differences were obsd. among the DE, KLD, and DEG groups. A slight dose-dependent increase of proliferative responses of mouse cervical lymph node cells to JCPAs in both DE and KLD groups was obsd., but not in the DEG group. Remarkable decrease of interferon-.gamma. and significant increase of interleukin-4 in the nasal lavage fluid were apparent after DE or DEG exposure, but not in the KLD group. These results suggest that these air pollutants (DE, KLD, and DEG) enhance the prodn. of IgE antibodies in mice, with similar adjuvant activities in each case. Furthermore, in the early phase of exposure in which sensitization occurred with exposure to pollen, the fine particles and gas components are considered to have exhibited different enhancing mechanisms in mice as follows: (1) The fine particles augmented prodn. of IgE antibodies through activation of T lymphocytes, and (2) the gas components exhibited almost no action on T lymphocytes, but directly induced disorders of the cytokine network and augmented the prodn. of IgE antibodies.

IT Air pollution
Airborne particles
Allergy
Cryptomeria japonica
Immunity
Immunotoxicity
Pollen
T cell (lymphocyte)
(diesel exhaust, kanto loam dust, or diesel exhaust without particles inhalation effect on immune responses in mice exposed to Japanese cedar (Cryptomeria Japonica) pollen)

IT **Allergens**
RL: ADV (Adverse effect, including toxicity); POL (Pollutant); BIOL (Biological study); OCCU (Occurrence)
(diesel exhaust, kanto loam dust, or diesel exhaust without particles inhalation effect on immune responses in mice exposed to Japanese cedar (Cryptomeria Japonica) pollen)

IT 7429-90-5, Aluminum, biological studies 7439-89-6, Iron, biological studies 7439-92-1, Lead, biological studies 7440-02-0, Nickel, biological studies 7440-42-8, Boron, biological studies 7440-47-3, Chromium, biological studies 7440-66-6, Zinc, biological studies 7440-70-2, **Calcium**, biological studies 7631-86-9, Silica, biological studies 7704-34-9, Sulfur, biological studies 7723-14-0, Phosphorus, biological studies
RL: ADV (Adverse effect, including toxicity); ANT (Analyte); POL (Pollutant); ANST (Analytical study); BIOL (Biological study); OCCU (Occurrence)

(in dust and particles; diesel exhaust, kanto loam dust, or diesel exhaust without particles inhalation effect on immune responses in mice exposed to Japanese cedar (Cryptomeria Japonica) **pollen**)

L8 ANSWER 4 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 2001:691713 CAPLUS
DN 135:240906
TI Method for **denaturing allergens** using **calcium** or **strontium** salts
IN Inui, Keiichiro; Mikame, Mariko
PA Sumitomo Chemical Co.,ltd., Japan; Shinto Fine Co., Ltd.
SO Eur. Pat. Appl., 14 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1133918	A1	20010919	EP 2001-105419	20010312
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2001328936	A2	20011127	JP 2001-56349	20010301
US 2001048097	A1	20011206	US 2001-802941	20010312
PRAI JP 2000-70918	A	20000314		

RE.CNT 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Method for **denaturing allergens** using **calcium** or **strontium** salts
AB A method is described for **denaturing allergerns**, esp. plant **allergens** and house dust **mite allergens**, using alk. earth metal salts such as **calcium** acetate, **calcium** nitrate, **calcium** iodide, **calcium** pantothenate, and **strontium** chloride.
ST **allergen denaturation calcium** **strontium** salt
IT **Allergens**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(Der f II (Dermatophagoides farinae, II); method for **denaturing allergens** using **calcium** or **strontium** salts)
IT Alcohols, biological studies
Alkaline earth salts
Polyoxyalkylenes, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(method for **denaturing allergens** using **calcium** or **strontium** salts)
IT **Allergens**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(method for **denaturing allergens** using **calcium** or **strontium** salts)
IT Acids, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(org.; method for **denaturing allergens** using **calcium** or **strontium** salts)
IT **Denaturation**
(protein; method for **denaturing allergens** using **calcium** or **strontium** salts)
IT Polymers, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)

(water-sol.; method for **denaturing allergens** using **calcium** or **strontium** salts)

IT 50-21-5, lactic acid, biological studies 50-81-7, ascorbic acid, biological studies 62-54-4, **calcium** acetate 64-19-7, acetic acid, biological studies 77-92-9, citric acid, biological studies 79-09-4, propionic acid, biological studies 87-69-4, tartaric acid, biological studies 89-65-6, isoascorbic acid 110-15-6, succinic acid, biological studies 110-16-7, maleic acid, biological studies 110-17-8, fumaric acid, biological studies 137-08-6, **calcium** pantothenate 140-99-8, **calcium** succinate 141-82-2, malonic acid, biological studies 299-28-5, **calcium** gluconate 471-34-1, **calcium** carbonate, biological studies 526-95-4, gluconic acid 814-80-2, **calcium** lactate 823-77-8, **calcium** nicotinate 3164-34-9, **calcium** tartrate, biological studies 4075-81-4, **Calcium** propionate 5793-94-2 6915-15-7, malic acid 7440-24-6D, **Strontium**, salts, biological studies 7440-70-2D, **Calcium**, salts, biological studies 7664-38-2, Phosphoric acid, biological studies 7732-18-5, water, biological studies 9002-89-5, Polyvinyl alcohol 9003-01-4, polyacrylic acid 9003-39-8, polyvinylpyrrolidone 9005-32-7, alginic acid 10043-52-4, **calcium** chloride, biological studies 10086-45-0, **calcium** pyrophosphate 10102-68-8, **calcium** iodide 10103-46-5, **calcium** phosphate 10124-37-5, **Calcium** nitrate 10476-85-4, **Strontium** chloride 17482-42-7, **calcium** malate 19455-76-6, **calcium** malonate 25322-68-3, polyethylene glycol 27214-00-2, **calcium** glycerophosphate 62624-30-0, ascorbic acid 65644-56-6, **calcium** glycerate
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(method for **denaturing allergens** using **calcium** or **strontium** salts)

L8 ANSWER 5 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 2001:608579 CAPLUS
DN 135:356490
TI Immunotherapy with a **calcium** phosphate-adsorbed five-grass-**pollen** extract in seasonal rhinoconjunctivitis: A double-blind, placebo-**controlled** study
AU Leynadier, F.; Banoun, L.; Dollois, B.; Terrier, P.; Epstein, M.; Guinnepain, M.-T.; Firon, D.; Traube, C.; Fadel, R.; Andre, C.
CS Hopital Rothschild, Paris, Fr.
SO Clinical and Experimental Allergy (2001), 31(7), 988-996
CODEN: CLEAEN; ISSN: 0954-7894
PB Blackwell Science Ltd.
DT Journal
LA English
RE.CNT 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI Immunotherapy with a **calcium** phosphate-adsorbed five-grass-**pollen** extract in seasonal rhinoconjunctivitis: A double-blind, placebo-**controlled** study
AB Background: **Calcium** phosphate-adsorbed **allergen** exts. are used for s.c. immunotherapy to avoid the use of aluminum adjuvants. Objectives: A double-blind, placebo-**controlled** study was performed to confirm the safety and assess the efficacy of a standardized five-grass-**pollen** ext. adsorbed onto **calcium** phosphate for specific immunotherapy (IT). Methods: Twenty-nine patients with seasonal rhinoconjunctivitis were randomized to receive either the active prepn. (16 patients) or placebo (13 patients), in a 1-yr study. During the increasing dose phase, an ext. ranging from 0.1 IR per mL to 50 IR per mL was administered at a rate of one s.c. injection per wk until a maintenance dose was reached. The patients were assessed by symptom diary

and rescue medications during seasonal exposure and specific nasal and skin reactivity before and after IT. Immunol. parameters (specific IgE and IgG4 antibodies) were assessed before, during and after IT. Results: The overall symptoms score (mean AUC) was not significantly different between the IT group and the placebo group during grass-pollen exposure (49.6 vs. 56, resp.). The total medication score (mean AUC) was significantly lower in the IT group than in the placebo group (11 vs. 41, $P < 0.01$, Mann-Whitney U-test). The cumulative symptom/medication score was significantly lower in the IT group than in the placebo group (64.5 vs. 102.3, $P < 0.05$, U-test). A significant increase in nasal reactivity threshold was obsd. after IT in the IT group (21.4 IR/mL before IT vs. 63.4 IR/mL after IT, $P < 0.01$, Wilcoxon), whereas no significant changes were obsd. in the placebo group (31.0 IR/mL before IT vs. 37.7 IR/mL after IT). IT induced a significant redn. in grass pollen cutaneous reactivity in the actively treated group ($P < 0.001$). A significant increase in serum-specific IgG4 antibody response was obsd. in the IT group (3.1% before IT vs. 10.1% after IT, $P < 0.001$). Nine patients in the IT group developed moderate immediate systemic reactions vs. two patients in the placebo group. Conclusion: Specific immunotherapy with **calcium** phosphate-adsorbed standardized grass **pollen** ext. was safe and effective for the treatment of patients with seasonal **allergic** rhinoconjunctivitis.

ST immunotherapy **calcium** phosphate adsorbed grass **pollen**; seasonal rhinoconjunctivitis antiallergy grass **allergen**

IT Immunoglobulins
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(E; immunotherapy with **calcium** phosphate-adsorbed five-grass-**pollen** ext. in seasonal rhinoconjunctivitis in humans)

IT Immunoglobulins
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(G4; immunotherapy with **calcium** phosphate-adsorbed five-grass-**pollen** ext. in seasonal rhinoconjunctivitis in humans)

IT Immunostimulants
(adjuvants; immunotherapy with **calcium** phosphate-adsorbed five-grass-**pollen** ext. in seasonal rhinoconjunctivitis in humans)

IT **Allergens**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(grass **pollen**; immunotherapy with **calcium** phosphate-adsorbed five-grass-**pollen** ext. in seasonal rhinoconjunctivitis in humans)

IT **Pollen**
(grass; immunotherapy with **calcium** phosphate-adsorbed five-grass-**pollen** ext. in seasonal rhinoconjunctivitis in humans)

IT **Allergy** inhibitors
Anthoxanthum odoratum
Hay fever
Immunotherapy
Lolium
Orchard grass
Timothy (Phleum pratense)
(immunotherapy with **calcium** phosphate-adsorbed five-grass-**pollen** ext. in seasonal rhinoconjunctivitis in humans)

IT Grass (Poaceae)
(meadow; immunotherapy with **calcium** phosphate-adsorbed five-grass-**pollen** ext. in seasonal rhinoconjunctivitis in humans)

IT Eye, disease
Nose
(rhinoconjunctivitis; immunotherapy with **calcium** phosphate-adsorbed five-grass-**pollen** ext. in seasonal rhinoconjunctivitis in humans)
IT 10103-46-5, **Calcium** phosphate
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(immunotherapy with **calcium** phosphate-adsorbed five-grass-**pollen** ext. in seasonal rhinoconjunctivitis in humans)

L8 ANSWER 6 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 2000:252944 CAPLUS
DN 132:278482
TI Medical food composition of **reduced allergenicity**, especially adapted for improving gut mucosal integrity
IN Liska, De Ann; King, Margaret; Medcalf, Darrell; Peterson, De Brian; Bland, Jeffrey
PA Healthcomm International, Inc., USA
SO U.S., 10 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 6051260	A	20000418	US 1998-56734	19980407

RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI Medical food composition of **reduced allergenicity**, especially adapted for improving gut mucosal integrity
IT Rice (Oryza sativa)
Rice (Oryza sativa)
(flour, parboiled; medical food compn. of **reduced allergenicity**, esp. adapted for improving gut mucosal integrity)
IT **Allergens**
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(medical food compn. of **reduced allergenicity**, esp. adapted for improving gut mucosal integrity)
IT Amino acids, biological studies
Canola oil
Carotenes, biological studies
Fructooligosaccharides
Vitamins
RL: FFD (Food or feed use); MOA (Modifier or additive use); BIOL (Biological study); USES (Uses)
(medical food compn. of **reduced allergenicity**, esp. adapted for improving gut mucosal integrity)
IT Intestine
(mucosa; medical food compn. of **reduced allergenicity**, esp. adapted for improving gut mucosal integrity)
IT Flours and Meals
Flours and Meals
(rice, parboiled; medical food compn. of **reduced allergenicity**, esp. adapted for improving gut mucosal integrity)
IT 50-81-7, Ascorbic acid, biological studies 52-89-1, L-Cysteine hydrochloride 56-85-9, L-Glutamine, biological studies 58-56-0, Pyridoxine hydrochloride 58-85-5, Biotin 58-95-7, .alpha.-Tocopheryl acetate 59-30-3, Folic acid, biological studies 67-03-8, Thiamine hydrochloride 67-97-0, Vitamin d3 68-19-9, Cyanocobalamin 70-18-8, Glutathione, biological studies 72-19-5, L-Threonine, biological studies 79-83-4, Pantothenic acid 83-88-5, Riboflavin, biological studies

98-92-0, Niacinamide 137-08-6, **Calcium** pantothenate 141-01-5, Ferrous fumarate 527-09-3, Copper gluconate 616-91-1, N-Acetylcysteine 6485-39-8, Manganese gluconate 7439-96-5, Manganese, biological studies 7439-98-7, Molybdenum, biological studies 7440-50-8, Copper, biological studies 7693-13-2, **Calcium** citrate 7757-93-9, Dicalcium phosphate 7758-11-4, Dibasic potassium phosphate 9004-54-0, Dextran, biological studies 9005-80-5, Inulin 10098-89-2, L-Lysine hydrochloride 11103-57-4, Vitamin a 12001-79-5, Vitamin k 17949-65-4, Zinc picolinate
RL: FFD (Food or feed use); MOA (Modifier or additive use); BIOL (Biological study); USES (Uses)
(medical food compn. of **reduced allergenicity**, esp. adapted for improving gut mucosal integrity)

L8 ANSWER 7 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 2000:72307 CAPLUS
DN 133:42065
TI Immunogold electron microscopic localization of the cross-reactive two-EF-hand **calcium**-binding birch **pollen** **allergen** bet v 4 in dry and rehydrated birch **pollen**
AU Grote, Monika; Hayek, Brigitte; Reichelt, Rudolf; Kraft, Dietrich; Valenta, Rudolf
CS Institute of Medical Physics and Biophysis, University of Munster, Munster, D-48149, Germany
SO International Archives of Allergy and Immunology (1999), 120(4), 287-294
CODEN: IAAIEG; ISSN: 1018-2438
PB S. Karger AG
DT Journal
LA English
RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI Immunogold electron microscopic localization of the cross-reactive two-EF-hand **calcium**-binding birch **pollen** **allergen** bet v 4 in dry and rehydrated birch **pollen**
AB Background: Recently, a novel family of low-mol.-wt. (8-9 kD), two-EF-hand **calcium**-binding proteins has been described as **allergens** in plant **pollens**. Approx. 10% of pollen-**allergic** patients have IgE antibodies which cross-react with the two-EF-hand **allergens** in tree, grass and weed pollens. The aim of the present study was to localize Bet v 4, the two-EF-hand **allergen** from birch, in mature, dry pollen and to study the release of this **allergen** after hydration of the pollen by immunogold electron microscopy. Methods: Using completely anhyd. fixation techniques in combination with immunogold electron microscopy, we localized Bet v 4 and, for **control** purposes, the major birch **pollen** **allergen** Bet v 1, in dry birch **pollen** as well as in **pollen** grains after different periods of hydration. Parallel with these morphol. studies, we monitored the release of Bet v 4 and Bet v 1 into aq. supernatants of hydrated birch pollen grains by immunoblotting. Results: Bet v 4 was found in the electron-dense cytosol, in particular between the vesicles and cisternae of the endoplasmic reticulum, inside mitochondria and in the vegetative as well as in the generative nucleus. Bet v 1 was localized in similar cellular compartments except for the mitochondria. After 30 s to 1 min of hydration, Bet v 4 migrated into the pollen exine and into the aq. supernatants. Bet v 1 also moved out of the pollen grain, though not as quickly as Bet v 4. Conclusion: Bet v 4 represents an intracellular pollen protein which, following hydration of pollen grains, rapidly migrates to the pollen surface (exine) and is washed out. This behavior explains how Bet v 4, being primarily an intracellular pollen protein, becomes available to sensitize patients.
ST pollen **allergen** betv4 IgE cytosol hydration
IT Immunoglobulins
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological

study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(E; **allergen** bet v 4 in dry and rehydrated birch pollen and its interaction with IgE)

IT **Allergy**
Cell nucleus
EF hand
Endoplasmic reticulum
Mitochondria
Pollen
(**allergen** bet v 4 in dry and rehydrated birch pollen and its interaction with IgE)

IT **Calcium**-binding proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**allergen** bet v 4 in dry and rehydrated birch **pollen** and its interaction with IgE)

IT **Allergens**
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(bet v 1 (Betula verrucosa, 1); **allergen** bet v 4 in dry and rehydrated birch pollen and its interaction with IgE)

IT **Allergens**
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(bet v 4 (Betula verrucosa, 4); **allergen** bet v 4 in dry and rehydrated birch pollen and its interaction with IgE)

L8 ANSWER 8 OF 59 CAPLUS COPYRIGHT 2002 ACS

AN 1998:466402 CAPLUS
DN 129:110226
TI Paints inhibiting the chitin synthesis in arthropods, for the **control** of pests and **allergens**
IN Mateo Herrero, Maria Pilar
PA Mateo Herrero, Maria Pilar, Spain
SO Eur. Pat. Appl., 4 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 851008	A2	19980701	EP 1997-500206	19971125
	EP 851008	A3	19981202		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	ES 2127120	A1	19990401	ES 1996-2723	19961223
	ES 2127120	B1	19991116		
	BR 9706291	A	19990518	BR 1997-6291	19971218
	US 5931994	A	19990803	US 1997-995132	19971219
PRAI	ES 1996-2723		19961223		

TI Paints inhibiting the chitin synthesis in arthropods, for the **control** of pests and **allergens**
ST paint chitin synthesis inhibitor; microencapsulation; arthropod pest **allergen control**
IT Acrylic polymers, uses
RL: TEM (Technical or engineered material use); USES (Uses)
(emulsions; paints inhibiting the chitin synthesis in arthropods, for the **control** of pests and **allergens**)

IT Acaricides
Arthropod (Arthropoda)
Insecticides
Paints
Pesticides
(paints inhibiting the chitin synthesis in arthropods, for the **control** of pests and **allergens**)

IT **Allergens**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(paints inhibiting the chitin synthesis in arthropods, for the **control** of pests and **allergens**)

IT Vinyl compounds, uses
RL: TEM (Technical or engineered material use); USES (Uses)
(polymers, emulsions; paints inhibiting the chitin synthesis in arthropods, for the **control** of pests and **allergens**)

IT Polyphosphoric acids
RL: TEM (Technical or engineered material use); USES (Uses)
(sodium salts; paints inhibiting the chitin synthesis in arthropods, for the **control** of pests and **allergens**)

IT 333-41-5, O,O-Diethyl O-(2-isopropyl-6-methylpyrimidin-4-yl) phosphorothioate 2921-88-2, Ethyl chlorpyriphos 35367-38-5, Diflubenzuron 41096-46-2, Hydroprene 52337-88-9, O,O-Diethyl O-(3,4,6-Trichloro-2-pyridyl) phosphorothioate 64628-44-0, Triflumuron 72490-01-8, Fenoxycarb 78587-05-0, Hexythiazox 86479-06-3, Hexaflumuron 101463-69-8, Flufenoxuron
RL: ARG (Analytical reagent use); TEM (Technical or engineered material use); ANST (Analytical study); USES (Uses)
(paints inhibiting the chitin synthesis in arthropods, for the **control** of pests and **allergens**)

IT 1398-61-4, Chitin
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(paints inhibiting the chitin synthesis in arthropods, for the **control** of pests and **allergens**)

IT 471-34-1, **Calcium** carbonate, uses 532-32-1, Sodium benzoate 7632-00-0, Sodium nitrite 13463-67-7, Titanium oxide, uses
RL: TEM (Technical or engineered material use); USES (Uses)
(paints inhibiting the chitin synthesis in arthropods, for the **control** of pests and **allergens**)

L8 ANSWER 9 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 1998:192093 CAPLUS
DN 128:191570
TI Two-site **allergen** immunoassay
IN Miller, Larry S.; Bhullar, Balwant S.; Tuttle, Richard S.; Moore, Victor S.
PA Procter and Gamble Co., USA
SO U.S., 21 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5731157	A	19980324	US 1993-175715	19931230

TI Two-site **allergen** immunoassay
AB An **allergen** immunoassay method features the use of a combination of (a) closely **controlled** (1) elevated temps. for assay reactions, (2) low temps. for reagents and samples, (3) times for assay steps and esp. assay reaction times, (4) reagent concns., and (5) reagent amts.; (b) the use of a fast and accurate method of sample prepn. that removes dust and contaminants; (c) the stabilization of samples to avoid auto- and antibody degrdn. and unwanted effects of sample contaminants; and (d) the formation of a colored product to det. the amt. of a specific **allergen**. This combination provides an assay that can be completed in a few hours while retaining the precision, accuracy, sensitivity and response curve of previous methods requiring much longer periods of time. The assay is esp. suitable for computer **control** using a robotic liq. distribution system and allows for the detn. of four

different specific **allergens** in one hundred sixty samples in duplicate with stds. and **controls** in an eight hour period with a significant redn. in the no. of steps and attended technician time over previous assays.

ST **allergen** immunoassay

IT **Allergens**
RL: ANT (Analyte); ANST (Analytical study)
(airborn; two-site **allergen** immunoassay)

IT IR radiation
(emitting substance; two-site **allergen** immunoassay)

IT Albumins, analysis
RL: ARU (Analytical role, unclassified); ANST (Analytical study)
(serum; two-site **allergen** immunoassay)

IT Affinity chromatography
Antimicrobial agents
Buffers
Chemiluminescent substances
Computer application
Detergents
Fluorescent substances
Immunoassay
Isotope indicators
Phosphorescent substances
Robotics
Sample preparation
Stabilizing agents
(two-site **allergen** immunoassay)

IT Enzymes, analysis
RL: ANT (Analyte); ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(two-site **allergen** immunoassay)

IT Antibodies
Reagents
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(two-site **allergen** immunoassay)

IT 9001-02-9, Carbohydrase 9001-92-7, Protease 9012-54-8, Cellulase 9014-01-1, Subtilisin 9035-73-8, Oxidase
RL: ANT (Analyte); ANST (Analytical study)
(two-site **allergen** immunoassay)

IT 330-13-2, P-Nitrophenyl phosphate 9000-81-1, Acetylcholinesterase 9001-37-0, Glucose oxidase 9001-78-9, Alkaline phosphatase 9002-13-5, Urease 9003-99-0, Peroxidase 9031-11-2
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(two-site **allergen** immunoassay)

IT 77-86-1, Tris buffer 7647-14-5, Sodium chloride, analysis 7772-98-7, Sodium thiosulfate 10043-52-4, **Calcium** chloride, analysis 26628-22-8, Sodium azide
RL: ARU (Analytical role, unclassified); ANST (Analytical study)
(two-site **allergen** immunoassay)

L8 ANSWER 10 OF 59 CAPLUS COPYRIGHT 2002 ACS

AN 1998:145077 CAPLUS

DN 128:242661

TI Homologous epitopes of model food major **allergens** from fish parvalbumins and egg white ovalbumin

AU Elsayed, Said

CS Allergy Research Group, Department of Clinical Biochemistry, University Hospital, University of Bergen, Norway

SO Progress in Allergy and Clinical Immunology, Proceedings of the International Congress of Allergology and Clinical Immunology, 16th, Cancun, Mex., Oct. 19-24, 1997 (1997), 127-131. Editor(s): Oehling, Albert K.; Huerta Lopez, J. G. Publisher: Hogrefe & Huber, Seattle, Wash. CODEN: 65SQAB

DT Conference; General Review
LA English
TI Homologous epitopes of model food major **allergens** from fish parvalbumins and egg white ovalbumin
AB A review with 35 refs. Fish and egg white **allergens** are among food **allergens** difficult to **eliminate**. **Allergen** M (Gad c I) is the major **allergen** of codfish, it belongs to muscle **calcium** binding proteins (parvalbumins), and it is a 12 kD protein composed of 113 amino acid residues (AA). Two **allergenically** active segments (PVA TM1 75 AA and PVA TM2 38 AA) are isolated by specific proteolytic cleavage, both of which are **allergenic** and immunogenic. PVA/TM2 is considered to be the smallest native **allergenic** fragment available from many fish species for exptl. immunotherapy (IT) and studies on cross immunogenicity of food **allergens**. Two synthetic peptides of **allergen** M, were extensively studied: peptide 49-64 of the CD-loop and peptide 88-103 of the EF-loop. The first Ca2+ binding peptide 49-64, binds specifically IgE and polyclonal IgG and is a consensus AA sequence found in more than 100 parvalbumins from many species. The second Ca2+ coordination loop (88-103) has been investigated in vivo and in vitro. Five major components namely ovalbumin OA (Gal d II), ovomucoid (Gal d I), ovotransferrin, ovomucin, and lysozyme represent about 80% of the total egg white protein amt. OA peptide 323-339 was investigated and shown to be immunogenic in rabbits and has the ability to bind to specific IgE. It has been also used in studies of T lymphocytes recognition of protein antigens and was on this basis suggested to be closely related to the peptide naturally created by APC during processing of OA. The same determinant is recognized by B cells and leads to specific IgE response. Peptide OA 323-339 is studied in many labs. and is a well documented B and T cell epitope of an **allergen**. OA 323-339, on the basis of extensive studies done by several labs., is extremely valuable tool for studying the mechanisms of human peptide-based IT.
ST parvalbumin ovalbumin epitope food **allergy** review
IT Egg white
Epitopes
Fish
(homologous epitopes of model food major **allergens** from fish parvalbumins and egg white ovalbumin)
IT **Allergens**
Ovalbumin
Parvalbumins
RL: PRP (Properties)
(homologous epitopes of model food major **allergens** from fish parvalbumins and egg white ovalbumin)

L8 ANSWER 11 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 1997:734918 CAPLUS
DN 128:33631
TI Immunological and biological properties of Bet v 4, a novel birch **pollen allergen** with two EF-hand **calcium** -binding domains
AU Engel, Edwin; Richter, Klaus; Obermeyer, Gerhard; Briza, Peter; Kungl, Andreas J.; Simon, Birgit; Auer, Manfred; Ebner, Christof; Rheinberger, Hans-Jorg; Breitenbach, Michael; Ferreira, Fatima
CS Inst. Genetik Allgemeine Biologie, Univ. Salzburg, Salzburg, A-5020, Austria
SO Journal of Biological Chemistry (1997), 272(45), 28630-28637
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
TI Immunological and biological properties of Bet v 4, a novel birch **pollen allergen** with two EF-hand **calcium**

-binding domains

AB The authors have isolated a cDNA clone coding for a birch pollen **allergen**, Bet v 4. The deduced amino acid sequence of Bet v 4 contained two typical EF-hand calcium-binding domains. Sequence similarities of Bet v 4 to calmodulin are primarily confined to the calcium-binding domains. However, significant sequence similarities extending outside the Ca2+-binding sites were found with a recently described group of pollen-specific **allergens** of Brassica and Bermuda grass. Both EF-hand domains of Bet v 4 are able to bind Ca2+, as demonstrated by 45Ca2+ blot overlay of wild type and calcium-binding deficient mutants of Bet v 4. Among pollen-**allergic** patients, protein-bound Ca2+ was not an abs. requirement for IgE recognition of Bet v 4. However, disruption of the carboxyl-terminal Ca2+-binding domain indicated that most IgE antibodies from **allergic** patients are directed against this site. IgE inhibition expts. suggested that Bet v 4 represents a highly cross-reactive pollen **allergen.** Pre-absorption of **allergic** sera with Bet v 4 drastically **reduced** IgE binding to proteins of similar mol. wt. in **pollen** exts. from distantly related plant species (e.g. timothy grass, mugwort, lily) but not in exts. from plant-derived foodstuff. To test for a possible biol. role in pollen germination and tube growth, the authors introduced recombinant Bet v 4 protein into growing, the authors introduced recombinant Bet v 4 protein into growing lily pollen tubes by iontophoresis. As a result, cytoplasmic streaming stopped in the vicinity of the electrode tip, and a slight depolarization of the membrane voltage was measured. These effects were not obsd. with Ca2+-binding deficient mutants of Bet v 4. Thus, Bet v 4 and homologous proteins represent a new class of pollen-specific Ca2+-binding **allergens** that may have a physiol. role as inhibitors of cytoplasmic streaming in outgrowing pollen tubes.

ST **allergen** Betv4 cDNA sequence birch pollen

IT **Allergens**
RL: PRP (Properties)
(Bet v 4 (Betula verrucosa, 4); **allergen** Bet v 4 of birch **pollen allergen** cDNA sequences, binding to **calcium** and human IgE, and role in **pollen** germination and tube growth)

IT Immunoglobulins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(E; **allergen** Bet v 4 of birch **pollen allergen** cDNA sequences, binding to **calcium** and human IgE, and role in **pollen** germination and tube growth)

IT Birch (Betula pendula)
Pollen
Pollen germination
(**allergen** Bet v 4 of birch **pollen allergen** cDNA sequences, binding to **calcium** and human IgE, and role in **pollen** germination and tube growth)

IT Gene, plant
RL: PRP (Properties)
(betv4; **allergen** Bet v 4 of birch **pollen allergen** cDNA sequences, binding to **calcium** and human IgE, and role in **pollen** germination and tube growth)

IT cDNA sequences
(for **allergen** Bet v 4 of birch pollen)

IT Protein sequences
(of **allergen** Bet v 4 of birch pollen)

IT 7440-70-2, **Calcium**, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**allergen** Bet v 4 of birch **pollen allergen** cDNA sequences, binding to **calcium** and human IgE, and role in

pollen germination and tube growth)

IT 198917-39-4
RL: PRP (Properties)
(amino acid sequence; **allergen** Bet v 4 of birch **pollen allergen** cDNA sequences, binding to **calcium** and human IgE, and role in **pollen** germination and tube growth)

IT 165765-56-0, GenBank X87153 199688-49-8, GenBank S54819
RL: PRP (Properties)
(nucleotide sequence; **allergen** Bet v 4 of birch **pollen allergen** cDNA sequences, binding to **calcium** and human IgE, and role in **pollen** germination and tube growth)

L8 ANSWER 12 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 1997:364155 CAPLUS
DN 127:45170
TI Glucocorticosteroids inhibit leukotriene production
AU Crocker, I. Caroline; Zhou, Chang Yi; Bewtra, Againdra K.; Kreutner, William; Townley, Robert G.
CS Creighton University Department of Medicine/Division of Allergy, Omaha, NE, USA
SO Annals of Allergy, Asthma, & Immunology (1997), 78(5), 497-505
CODEN: ALAIF6; ISSN: 1081-1206
PB American College of Allergy, Asthma, & Immunology
DT Journal
LA English
AB The mode of action of corticosteroids, important drugs in the treatment of inflammatory disease, is not yet fully understood. Corticosteroids are known to inhibit phospholipase A2 in unprimed eosinophils and basophils, preventing leukotriene synthesis, but their effect on cells that are already primed is unknown. As inflammatory cells from atopic subjects are often primed in vivo, the authors studied the effects of two potent corticosteroids on basophil sulfidoleukotriene prodn. in peripheral blood mixed leukocytes (PBML) from in-season and out-of-season atopic individuals. Cells were incubated for 24 h with mometasone furoate or beclomethasone dipropionate, primed with IL-3, stimulated with **calcium** ionophore, buffer, **allergen** or anti-IgE, and leukotriene prodn. was quantified. Peripheral blood mononuclear leukocytes from five of ten donors (in season) produced elevated sulfidoleukotrienes without a stimulus; cells from seven donors responded to anti-IgE by increased sulfidoleukotrienes. Neither steroid consistently affected sulfidoleukotriene prodn. in anti-IgE-stimulated cells which were releasing sulfidoleukotrienes in the absence of a stimulant. In comparison, sulfidoleukotriene prodn. was significantly **reduced** by 0.01 to 10 nM beclomethasone dipropionate or mometasone furoate when the cells were primed with IL-3 after exposure to the drug and stimulated with **calcium** ionophore or **allergen**, but no dose-relationship was apparent. Leukotriene prodn. by PBML in response to anti-IgE was potently inhibited by all concns. of mometasone furoate (0.01 nM to 1 .mu.M) with an inhibitory concn.50 of less than 0.01 nM. Beclomethasone dipropionate inhibited sulfidoleukotriene prodn. in this group (inhibitory concn.50 6 nM) in a dose-dependent manner. Sulfidoleukotriene prodn. and, conceivably, priming may be more effectively inhibited by mometasone furoate than beclomethasone dipropionate.

L8 ANSWER 13 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 1995:711728 CAPLUS
DN 123:110026
TI **Allergen**-stimulated interleukin-4 and interferon-.gamma. production in primary culture: responses of subjects with **allergic** rhinitis and normal **controls**

AU Imada, M.; Estelle, F.; Simons, R.; Jay, F. T.; Hayglass, K. T.
CS Departments Immunology, Pediatrics and Medical Microbiology, University Manitoba, Winnipeg, Can.
SO Immunology (1995), 85(3), 373-80
CODEN: IMMUAM; ISSN: 0019-2805
PB Blackwell
DT Journal
LA English
TI **Allergen**-stimulated interleukin-4 and interferon-.gamma. production in primary culture: responses of subjects with **allergic** rhinitis and normal **controls**
AB The balance of interleukin-4 (IL-4) to interferon-.gamma. (IFN-.gamma.) prodn. that is induced following exposure to common environmental antigens is believed to be instrumental in detg. whether hypersensitivity or clin. unresponsiveness results to that antigen. To date, evaluation of cytokine (protein) prodn. has been based predominately on **allergen** -reactive CD4 T-cell clones or activation of fresh unselected peripheral blood mononuclear cell (PBMC) populations with non-physiol. stimuli such as phorbol myristate acetate (PMA) and **calcium** ionophore, phytohemagglutinin (PHA), anti-CD3 or anti-CD2/anti-CD28 monoclonal antibodies (mAb). Here, ultrasensitive IL-4 and IFN-.gamma. assays were optimized to allow direct anal. of antigen-stimulated cytokine prodn. by fresh human PBMC. Primary cultures of cells from grass **pollen** -sensitive **allergic** rhinitis subjects and non-atopic **controls** were stimulated using a range of grass **pollen** **allergen** concns. in the absence of exogenous cytokines or polyclonal activators. The majority of subjects (45 to 52) exhibited chloroquine-sensitive, CD4-dependent cytokine prodn. in **allergen** -stimulated, short-term primary culture. Median IL-4 prodn. was substantially greater among atopics (13.0 pg/mL vs. < 1 pg/mL, Mann-Whitney U test, $P < 0.000001$) and IFN-.gamma. was lower ($P = 0.008$), providing direct evidence for an imbalance in both IL-4 and IFN-.gamma. prodn. among circulating, pollen-reactive cells in individuals with seasonal **allergic** rhinitis. The distinction in the **allergen**-driven cytokine responses elicited from normal and atopic donors was underscored by examn. of the ratios of IFN-.gamma.: IL-4 synthesis. Non-atopic individuals exhibited intense IFN-.gamma. dominance of the T-cell response, in marked contrast to that obsd. among grass pollen-sensitive individuals (median IFN-.gamma.: IL-4 ratios of 14.0 vs. 0.096, $P = 0.000002$). The observation that essentially all individuals produced IFN-.gamma. (.+-.IL-4) following antigen stimulation in vitro argues that the most relevant consideration in detg. susceptibility to immediate hypersensitivity vs. clin. tolerance to environmental **allergens** is not a genetically defined capacity to recognize the antigen (i.e. if **allergen**-reactive T cells are present in that individual) but the nature of the cytokine response.
ST **allergy** interleukin 4 interferon gamma
IT **Allergy**
Hay fever
(**allergen**-stimulated interleukin-4 and interferon-.gamma. prodn. by mononuclear leukocytes from humans with **allergic** rhinitis)
IT **Allergens**
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(**allergen**-stimulated interleukin-4 and interferon-.gamma. prodn. by mononuclear leukocytes from humans with **allergic** rhinitis)
IT Immune tolerance
(**allergen**-stimulated interleukin-4 and interferon-.gamma. prodn. by mononuclear leukocytes from humans with **allergic** rhinitis in relation to)
IT Lymphokines and Cytokines
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL

(Biological study); FORM (Formation, nonpreparative)
(interleukin 4, **allergen**-stimulated interleukin-4 and interferon-.gamma. prodn. by mononuclear leukocytes from humans with **allergic** rhinitis)

IT Interferons
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(.gamma., **allergen**-stimulated interleukin-4 and interferon-.gamma. prodn. by mononuclear leukocytes from humans with **allergic** rhinitis)

L8 ANSWER 14 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 1995:514043 CAPLUS
DN 122:263428
TI Production of diacylglycerol and arachidonic acid in peripheral blood mononuclear cells from patients with asthma and healthy controls
AU Dooper, Marten W S M.; Timmermans, Adiet; Aalbers, Rene; Weersink, Els J M.; de Monchy, Jan G R.; Kauffman, Henk F.
CS Clinic for Internal Medicine, State University Hospital, Groningen, 9713 EZ, Neth.
SO Annals of Allergy, Asthma, & Immunology (1995), 74(3), 248-54
CODEN: ALAIF6; ISSN: 1081-1206
DT Journal
LA English
AB Enhanced activities of peripheral blood cells are a common characteristic of patients with asthma. Here the authors tested whether this could be due to a dysfunction in .gtoreq.1 signal transduction systems. The prodn. of 1,2-diacylglycerol (1,2-DAG) and arachidonic acid was compared in mononuclear blood cells from patients with asthma and healthy controls. Using 3 different stimuli (Con A, aluminum fluoride, or the **calcium** ionophore A23187) no difference in the prodn. of both 1,2-DAG and arachidonic acid could be found between patients and **controls** before **allergen** challenge. Con A-induced 1,2-DAG prodn. could be inhibited completely in the presence of isoprenaline; Con A-induced arachidonic acid prodn., partially. The inhibitory effect of adenylate cyclase activation on the prodn. of 1,2-DAG and arachidonic acid was identical in patients and controls. Following **allergen** challenge, there was a tendency to an increased prodn. of 1,2-DAG and arachidonic acid in **controls**, whereas in patients there was a tendency to a decreased prodn. Thus, enhanced cellular activities found in patients with asthma are not caused by an intrinsic dysfunction in prodn. of 1,2-DAG and arachidonic acid.

L8 ANSWER 15 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 1994:101269 CAPLUS
DN 120:101269
TI Colorimetric method and reagents for detecting pollen in the air
IN Suzuki, Masahiro; Nomoto, Yoichi; Akaike, Toshihiro
PA Ekuosu Risaachi Kk, Japan
SO Jpn. Kokai Tokkyo Koho, 6 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 05284995	A2	19931102	JP 1992-116870	19920409
JP 2724939	B2	19980309		

AB The method comprises (1) exposing the cellular content (i.e. peroxidase) of any pollen in the (air) sample, (2) contacting the cellular content with H2O2 and a chromogen, and (3) measuring the color or absorbance. The method is simple and accurate in detg. air pollen, and is useful for pollen **allergy** prevention. Thus, a sample contg. Cryptomeria

pollen was detd. by mixing the sample with a soln. contg. 4-aminoantipyrine 15 mg, p-chlorophenol 15 mg, 0.3% H2O2 1 mL, and **CaCl2** 3 gm in a mortar, crushing the mixt. using a pestle, and measuring the absorbance at 505 nm.

IT **Pollen**
(detn. of, in air, test reagent contg. aminoantipyrine and chlorophenol and peroxide and **calcium** chloride for)

IT Air analysis
(**pollen** detection in, test reagent contg. aminoantipyrine and chlorophenol and peroxide and **calcium** chloride for)

IT Cryptomeria
(**pollen**, detn. of, in air, test reagent contg. aminoantipyrine and chlorophenol and peroxide and **calcium** chloride for)

IT **Allergy**
(to pollen, prevention of, air pollen test reagent in relation to)

IT Size **reduction** apparatus
(pestles, exposure of **pollen** peroxidase with, for **pollen** detn. in air)

IT 9003-99-0, Peroxidase
RL: ANST (Analytical study)
(**pollen**, detection of, colorimetric test reagent contg. aminoantipyrine and chlorophenol and peroxide and **calcium** chloride for)

IT 83-07-8, 4-Aminoantipyrine 106-48-9, p-Chlorophenol 7722-84-1, Hydrogen peroxide, uses 10043-52-4, **Calcium** chloride, uses
RL: ANST (Analytical study)
(test reagent contg., for detecting **pollen** in air)

L8 ANSWER 16 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 1994:29832 CAPLUS
DN 120:29832
TI **Allergen-reduced** rice, manufacture of the rice by treatment with aqueous salt solutions, and rice products made from the rice
IN Ikezawa, Yoshiro; Nishio, Takeshi; Iida, Shuichi; Tsubaki, Kazufumi; Suzuki, Takashi
PA Norinsuisansho Nogyo Seibutsu, Japan; Asahi Denka Kogyo Kk
SO Jpn. Kokai Tokkyo Koho, 8 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 05236889	A2	19930917	JP 1992-32744	19920123
JP 3055729	B2	20000626		

TI **Allergen-reduced** rice, manufacture of the rice by treatment with aqueous salt solutions, and rice products made from the rice

AB Rice, in which proteins with mol. wt. 12,000-30,000, 30,000-40,000, and 50,000-60,000 are practically removed, is manufd. by treatment of glutelin- and/or prolamin-low rice with aq. salt solns. Low-glutelin-rice was stirred with 1M NaCl contg. MO 750 (decaglycerin monooleate) and Protease N "Amano" (protease) at 10.degree. for 12 h, centrifuged, the procedure was repeated twice, the ppt. was stirred with H2O for 2 h, and the ppt. was dried to manuf. low-**allergen** rice, which did not cause **allergy** in rice **allergy** patients.

ST rice **allergen** protein removal salt; glutelin low rice **allergen** removal; prolamin low rice **allergen** removal

IT Proteins, biological studies
RL: BIOL (Biological study)
(**allergens**, in glutelin- and/or prolamin-low rice, removal

of, with aq. salt solns.)

IT Salts, uses
RL: USES (Uses)
(aq. solns. contg., protein **allergens** removal from glutelin- and/or prolamin-low rice with)

IT Rice
(glutelin and/or prolamin-low, protein **allergens** removal from, with aq. salt solns.)

IT **Allergens**
RL: BIOL (Biological study)
(proteins, in glutelin- and/or prolamin-low rice, removal of, with aq. salt solns.)

IT Glutelins
Prolamins
RL: BIOL (Biological study)
(rice low in, protein **allergens** removal from, with aq. salt solns.)

IT Bakery products
(crackers, rice, contg. glutelin- and/or prolamin-low rice with **reduced** levels of protein **allergens**)

IT 7647-14-5, Sodium chloride, biological studies 7757-82-6, Sodium sulfate, biological studies 10043-52-4, **Calcium** chloride, biological studies
RL: BIOL (Biological study)
(aq. solns. contg., protein **allergens** removal from glutelin- and/or prolamin-low rice with)

L8 ANSWER 17 OF 59 CAPLUS COPYRIGHT 2002 ACS

AN 1990:75605 CAPLUS

DN 112:75605

TI Hypoallergenic low-.beta.-lactoglobulin whey preparations

IN De Rham, Olivier

PA Societe des Produits Nestle S. A., Switz.

SO Eur. Pat. Appl., 6 pp.
CODEN: EPXXDW

DT Patent

LA French

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 311795	A1	19890419	EP 1988-114870	19880912
	EP 311795	B1	19911204		
	R: AT, BE, DE, ES, FR, GB, GR, IT, LU, NL, SE				
	CH 672230	A	19891115	CH 1987-4041	19871015
	AT 69927	E	19911215	AT 1988-114870	19880912
	ES 2027744	T3	19920616	ES 1988-114870	19880912
	US 4879131	A	19891107	US 1988-245463	19880916
	CA 1322688	A1	19931005	CA 1988-579494	19881006
	JP 01132335	A2	19890524	JP 1988-257452	19881014
	JP 07046966	B4	19950524		
PRAI	CH 1987-4041		19871015		
	EP 1988-114870		19880912		

AB The content of the **allergenic** .beta.-lactoglobulins of whey is **reduced** to (0.1% by heating in the presence of Ca2+ to ppt. the protein. Demineralized sweet whey was adjusted to 10% solids and 0.8% whey protein added. The pH was adjusted to 6.35 and the free Ca2+ to 13 mM followed by heating to 95.degree. for 10 min, followed by rapid cooling. The whey content of the liq. phase was reduced to 0.05% of its original value. Of eight guinea pigs fed with this prepn. only two showed development of an **allergic** response to whey.

IT **Allergens**
RL: BIOL (Biological study)
(lactoglobulin, removal from whey of)

IT 10043-52-4, **Calcium** chloride, biological studies
RL: BIOL (Biological study)
(**allergenic** lactoglobulin removed from whey by heating with)

L8 ANSWER 18 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 1975:135673 CAPLUS
DN 82:135673
TI Pollen-wall proteins. Physicochemical characterization and role in self-incompatibility in Cosmos bipinnatus
AU Howlett, B. J.; Knox, R. B.; Paxton, J. D.; Heslop-Harrison, J.
CS Bot. Dep., Aust. Natl. Univ., Canberra, Aust.
SO Proc. R. Soc. London, Ser. B (1974), 188(1091), 167-82
CODEN: PRLBA4
DT Journal
LA English
AB Fresh **pollen** of C. bipinnatus was extd. with an isotonic mannitol-**CaCl2** medium that preserved **pollen** viability. The pollen-wall diffusates after partial purifn. by (NH4)2SO4 pptn. and gel filtration contained > 7 protein bands. Two fractions contained demonstrable carbohydrate, suggesting they are glycoproteins. After sodium dodecyl sulfate gel electrophoresis, many bands were obtained, the 2 major fractions having estd. (relative) mol. masses of 11,500 and 30,000. Gel patterns of C. bipinnatus pollen diffusate were compared with those from ragweed diffusate and antigen E. The **pollen**-wall proteins were implicated in the **control** of self incompatibility. In compatible matings, most pollen tubes had grown through the style to the ovary within 60 min after pollination. After self pollination, the pollen tube was arrested on the stigma surface, and the callose rejection response was detected within 15 min of pollination. Self incompatiblity was partially overcome (to .apprx.27% of compatible seed set) with pollen mixes of killed compatible and fresh self pollen. These could be replaced with equal effect by applying compatible pollen-wall diffusate (contg. 1 mg/ml protein) followed by self pollination. The active proteins were heat stable, and included an antigen E-like fraction with partial immunol. identity to the ragweed **allergen**.

L8 ANSWER 19 OF 59 CAPLUS COPYRIGHT 2002 ACS
AN 1962:438778 CAPLUS
DN 57:38778
OREF 57:7786f-i
TI The chemistry of **allergens**. **Inactivation** of the castor bean **allergens** and ricin by heating with aqueous **calcium** hydroxide
AU Spies, Joseph R.; Coulson, E. J.; Bernton, Harry S.; Wells, P. A.; Stevens, Henry
CS U.S. Dept. of Agr., Washington, DC
SO J. Agr. Food Chem. (1962), 10, 140-4
DT Journal
LA Unavailable
TI The chemistry of **allergens**. **Inactivation** of the castor bean **allergens** and ricin by heating with aqueous **calcium** hydroxide
AB cf. CA 37, 54831, 67384; 38, 37174; 46, 7523e; 54, 21425b. The conditions of time, temp., and pH for the **inactivation** with Ca(OH)2 of 2 principal harmful components of castor beans (an unusually stable **allergen** and ricin, a less stable, extremely toxic protein) were detd. A safe-to-handle mixt. of pomace and CaHPO4 resulted. A relation between destruction of the immune-pptg. and reagin-neutralizing properties of the **allergen** was observed. At pH 5.9 to 8.7, heating destroyed the reagin-neutralizing property before the pptg. property, but at pH 10.8 to 11.9 the reverse was observed. Castor beans yield .apprx.50% of oil and pomace each. The pomace utilized as fertilizer entails some hazard because of its residual **allergen** content.

Enhancement of the market value of the castor bean crop is predicted if **inactivation** of the **allergens** extends the usefulness of the pomace as a fertilizer or livestock feed. 19 references.

IT Castor beans
(**allergens** and ricin of, Ca(OH)2 **inactivation** of)

IT **Allergens**
(of castor bean, Ca(OH)2 **inactivation** of)

IT 1305-62-0, **Calcium** hydroxide
(**allergens** and ricin in castor beans in relation to)

L8 ANSWER 20 OF 59 WPIDS (C) 2002 THOMSON DERWENT
AN 2002-444948 [48] WPIDS
CR 2002-454488 [48]; 2002-489748 [52]
DNN N2002-350540 DNC C2002-126776
TI **Allergen** neutralization composition for inanimate objects, comprising preset amount of **allergy** neutralizing aluminum ion and solvent, is sprayable such that preset amount of aluminum ion is provided as aluminum sulfate.
DC C07 D22 E19 E33 E35 E37 P34
IN CASTRO, M B; CHATTERJEE, R; KOBAYASHI, R; LI, Y; OH, H; YOSHIKAWA, A
PA (PROC) PROCTER & GAMBLE CO
CYC 2
PI CA 2357839 A1 20020329 (200248)* EN 37p
AU 2001077324 A 20020411 (200248)
ADT CA 2357839 A1 CA 2001-2357839 20010927; AU 2001077324 A AU 2001-77324 20010928
PRAI US 2001-311634P 20010810; WO 2000-US27018 20000929; WO 2000-US27019 20000929; WO 2001-US4070 20010208
TI **Allergen** neutralization composition for inanimate objects, comprising preset amount of **allergy** neutralizing aluminum ion and solvent, is sprayable such that preset amount of aluminum ion is provided as aluminum sulfate.
AB CA 2357839 A UPAB: 20020916
NOVELTY - An **allergen** neutralization composition (ANC), comprises **allergy** neutralizing aluminum ion (0.01-1.0 weight% (wt.%), preferably 0.10-0.50 wt.%), and a solvent. ANC is sprayable such that at least 85 weight% (wt.%), preferably at least 98 wt.% of aluminum ion is provided as aluminum sulfate.

USE - For use on inanimate objects, for **controlling allergen** containing dust particles. ANC suppresses **allergen** compounds, particularly the **allergens** associated with house dust **mites** and other common **allergens** such as cat dander, cockroaches and **pollen**. ANC is sprayed onto household surfaces such as counter tops, cabinets, walls, floors, bathroom surfaces and kitchen surfaces. A mist of the composition is sprayed onto fabric and/or fabric articles including clothes, curtains, drapes, upholstered furniture, carpeting, bed lines, bath lines, table-cloths, sleeping bags, tents, car interior, etc. Also sprayed onto cat litter, pet bedding and pet houses.

ADVANTAGE - ANC **controls allergen** containing dust particles without leaving behind a sticky feeling on household surfaces. ANC provides superior performance in **reducing** consumer's **allergy** symptoms. The compositions operate on the principle of neutralizing the proteins associated with common house dust **mites**, cockroaches, cats and **pollen**, without killing the house dust **mites**. The proteins can be neutralized chemically by **denaturing**, or they can be physically disabled. The proteins that cause **allergic** reactions in humans are neutralized or kept from entering the human body. The compositions in addition to providing improved efficacy, are compatible with a wide variety of household surfaces. Aluminum ions function as excellent **allergen** neutralization compound, when the aluminum ion is supplied as a salt of sulfate. Additional **allergen denaturing** compounds such

as low molecular alcohol ensures solubility and stability of the **allergen denaturing** compounds.
Dwg.0/0

TECH UPTX: 20020730
TECHNOLOGY FOCUS - ORGANIC CHEMISTRY - Preferred Components: The composition comprises no aluminum chloro hydrate and further comprises a wetting agent and miticide. The additional **allergen denaturing** compounds is selected from polyphenol compounds, hydrogen peroxide, salicyclic acid, citric acid, lactic acid, glycolic acid, ascorbic acid, gallic acid, gluconic acids and additional metal ions. The additional metal ions are zinc, stannous, stannic, magnesium, **calcium**, manganese, titanium, copper and/or nickel, preferably the additional metal ion is zinc and/or stannous. The solvent comprises water. Preferred Properties: ANC neutralizes at least 40 wt.%, preferably at least 90% of **allergen** containing proteins as measured by ELISA test protocol. Preferred Amount: The composition comprises less than 10 wt.%, preferably less than 1 wt.% of the aluminum ion is provided as aluminum chlorohydrate. The solvent comprises 0.01-20 wt.%, preferably 0.1-5.0 wt.% of a volatile lower alcohol. Preferred Mechanism: ANC is sprayed on dust particles, the particles tend to agglomerate such that the medium particle size of the dust particles increases by at least 20 wt.%, preferably at least 30 wt.%, from the median particle size of dust sprayed with a compositionally equivalent solution that comprises no aluminum ions.

TT TT: **ALLERGEN** NEUTRALISE COMPOSITION INANIMATE OBJECT COMPRISE PRESET AMOUNT **ALLERGIC** NEUTRALISE ALUMINIUM ION SOLVENT SPRAY PRESET AMOUNT ALUMINIUM ION ALUMINIUM SULPHATE.

L8 ANSWER 21 OF 59 WPIDS (C) 2002 THOMSON DERWENT
AN 2002-305681 [35] WPIDS
DNC C2002-089095
TI Medicine for preventing and treating **allergic** bronchial asthma and its preparation.
DC B04
IN WANG, D; WANG, S
PA (WANG-I) WANG S
CYC 1
PI CN 1335149 A 20020213 (200235)*
ADT CN 1335149 A CN 2000-121446 20000724
PRAI CN 2000-121446 20000724
TI Medicine for preventing and treating **allergic** bronchial asthma and its preparation.
AB CN 1335149 A UPAB: 20020603
NOVELTY - The medicine for preventing and treating **allergic** bronchial asthma is prepared with theine, amobarbital, mephedrine, chlorphenamine, **calcium** hydrogen phosphate, dexamethasone, vitamin D2, amur lilac extract and starch and through crushing, sieving, mixing, preparation of adhesive, pelletizing and finishing. The medicine comprising both Western and Chinese medicine components can **control allergic** bronchial asthma caused by cold air, **pollen**, environmental pollution, pesticide, chemical fertilizer, etc. and has also obvious preventing and treating effect on asthma of bronchitis, pulmonary emphysema, infant pneumonia sequelae.
Dwg.0/0
TT TT: MEDICINE PREVENT TREAT **ALLERGIC** BRONCHIAL ASTHMA PREPARATION.

L8 ANSWER 22 OF 59 WPIDS (C) 2002 THOMSON DERWENT
AN 1997-172993 [16] WPIDS
DNC C1997-055215
TI Soybean protein contg. low **allergen** - is prepd. by removing GLy mI from base unit having no alpha sub-units, useful as food additives.
DC B04 D13

PA (FUKO) FUJI SEIYU KK; (NORQ) NORINSUISANSHO TOHOKUNOGYO SHIKENJYO CHO
CYC 1
PI JP 09037720 A 19970210 (199716)* 6p
ADT JP 09037720 A JP 1995-195652 19950801
PRAI JP 1995-195652 19950801
TI Soybean protein contg. low **allergen** - is prepd. by removing GLy mI from base unit having no alpha sub-units, useful as food additives.
AB JP 09037720 A UPAB: 19970417
Soybean protein contg. low **allergen** is prepd. by removing Gly mI from base protein contg. no alpha subunits. Also claimed is a low-**allergen** soybean protein prepd. from base soybean contg. no alpha-subunit. Soybean protein extracted from base contg. no alpha subunit is treated using acidic aq. soln. of pH 3.5-5 contg. 90 mM or more of acetic anion, and 1200mM or more of chlorine ion, or using acidic aq. soln. of pH 2-4 , contg. 3mM or more of polybasic acid or acetic acid or 600 mM or more of chlorine ion for selective precipitation of Gly mI to give the supernatant contg. low-**allergen** soybean protein. Supernatant is treated in electric **reduction**, or using **reducing** agents and purified.

USE - Low-**allergen** soybean protein are used as food additives soybean **allergic** diseases.

In an example, skimmed soybean (100g) contg. no alpha`, and alpha subunit was extracted in water (1500ml) contg. 1N NaOH at room temp. for 3 hrs. to give soybean milk. **CaCl2** (30 mM or 40 mM) was added to soybean milk, stirred in addn. of 2N sulphuric acid for pH of 2.8, and centrifuged to give supernatant, which was purified for removal of whey-protein in isoelectro precipitation, and electrophoresis to give low-**allergen** protein.

Dwg.0/4
TT TT: SOY PROTEIN CONTAIN LOW **ALLERGEN** PREPARATION REMOVE BASE UNIT NO ALPHA SUB UNIT USEFUL FOOD ADDITIVE.

L8 ANSWER 23 OF 59 WPIDS (C) 2002 THOMSON DERWENT
AN 1992-339007 [41] WPIDS
DNN N1992-258568 DNC C1992-150810
TI Diagnosing **allergy** - by contacting blood sample with **allergen**, introducing fluorescent **calcium** colourant and measuring fluorescence intensity.
DC B04 S03
IN KIRILLOV, M A; NISHEVA, E S; VORONTSOV, I M
PA (LEPE-R) LENGD PEDIATRIC MEDICINE INST
CYC 1
PI SU 1691751 A1 19911115 (199241)* 3p
ADT SU 1691751 A1 SU 1988-4604078 19881109
PRAI SU 1988-4604078 19881109
TI Diagnosing **allergy** - by contacting blood sample with **allergen**, introducing fluorescent **calcium** colourant and measuring fluorescence intensity.
AB SU 1691751 A UPAB: 19931115
The method comprises incubation of leucocytes of peripheral blood with an **allergen**, and measuring **calcium** content in cells using a fluorescent **calcium** dye. To improve the accuracy and accelerate the method, granulocytes sepd. from the blood are used as samples, and 'Queen-2' is used as the fluorescent **calcium**-colouring agent, in concn. 500 mM. Fluorescence intensity is measured using cytofluorimeter. If the fluorescence intensity is increased by 10% w.r.t. **control** sample contg. no **allergen**, the patient is **allergic** to the **allergen**. Granulocytes are used at concn. 2x10 power 6/ml.

USE/ADVANTAGE - In medicine as a method of diagnosing **allergies** in patients. The proposed method ensures high accuracy of diagnosis (by 50% higher than that of the known method), **reduces** amt. of blood needed for analysis by 15 times, amt. of required **allergen** by 6 times and the duration of analysis by

half. Bul.42/15.11.91
Dwg. 0/0
TT TT: DIAGNOSE **ALLERGIC** CONTACT BLOOD SAMPLE **ALLERGEN** INTRODUCING FLUORESCENT **CALCIUM** COLOUR MEASURE FLUORESCENT INTENSITY.

L8 ANSWER 24 OF 59 WPIDS (C) 2002 THOMSON DERWENT
AN 1985-030505 [05] WPIDS
DNC C1985-013065
TI Milk prodn. for people **allergic** to cows milk - by diluting original milk with a calcium chloride soln., bringing to boil, cooling and removing residues.
DC D13
IN GURGENIDZE, G V; MONIAVA, I I
PA (TBIL-R) TBILISI MED INST
CYC 1
PI SU 1101214 A 19840707 (198505)* 4p
ADT SU 1101214 A SU 1980-2966039 19800801
PRAI SU 1980-2966039 19800801
TI Milk prodn. for people **allergic** to cows milk - by diluting original milk with a calcium chloride soln., bringing to boil, cooling and removing residues.
AB SU 1101214 A UPAB: 19930925
The original milk is boiled, cooled, and the precipitated residue in the form of proteins is removed. The **allergenicity** of the milk is **reduced** by removing the globulins and part of the casein, by introducing into the natural cow's milk, prior to boiling, a soln. of **calcium** chloride to achieve a concn. of 0.03-0.1%, with the precipitated residue of the proteins being sepd. by filtration or centrifuging.
USE/ADVANTAGE - In milk processing, to reduce the effects it has on people who are **allergic** to ordinary cow's milk.
0/5
TT TT: MILK PRODUCE PEOPLE **ALLERGIC** COW MILK DILUTE ORIGINAL MILK CALCIUM CHLORIDE SOLUTION BOIL COOLING REMOVE RESIDUE.

L8 ANSWER 25 OF 59 WPIDS (C) 2002 THOMSON DERWENT
AN 1976-96172X [51] WPIDS
TI Deodorant and antiperspirant compsn. - comprising zinc oxide, phenol, glycerin and calcium hydroxide in cream base.
DC B05 D21 D22 E12 E17
PA (STAF-I) STAFFIER D T
CYC 1
PI US 3996346 A 19761206 (197651)*
PRAI US 1975-559548 19750318; US 1976-664132 19760305
AB US 3996346 A UPAB: 19930901
A cream deodorant and antiperspirant compsn. comprises 12-50 wt. % zinc oxide, 0.1-0.4% phenol, 3.18% glycerin, 0.1-9.0% **calcium** hydroxide, and 30-94% of a cream base, a predetermined portion of the ZnO and phenol being in the form of Zn phenate. The compsn. **reduces** perspiration and body odour, but has low toxicity and is non-**allergenic**.

L8 ANSWER 26 OF 59 MEDLINE
AN 2002395772 IN-PROCESS
DN 22139544 PubMed ID: 12144561
TI A new oligomeric parvalbumin **allergen** of Atlantic cod (Gad mI) encoded by a gene distinct from that of Gad cI.
AU Das Dores S; Chopin C; Villaume C; Fleurence J; Gueant J-L
SO ALLERGY, (2002) 57 Suppl 72 79-83.
Journal code: 7804028. ISSN: 0105-4538.
CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)

LA English
FS IN-PROCESS; NONINDEXED; Priority Journals
ED Entered STN: 20020730
Last Updated on STN: 20020730
TI A new oligomeric parvalbumin **allergen** of Atlantic cod (Gad mI) encoded by a gene distinct from that of Gad cI.
AB BACKGROUND: The major **allergen** of Baltic cod (Gadus callarias) is a 12.3-kDa parvalbumin with two **calcium**-binding sites corresponding to EF-hand motifs. Our group found a 24-kDa IgE-reactive band that was also recognized by a monoclonal antiparvalbumin antibody in Atlantic cod (Gadus morhua). Our purpose was to purify and to determine the cDNA deduced sequence of this new cod **allergen**. METHODS: Proteins from pre rigor mortis Atlantic cod were separated by gel filtration and the eluted peaks were analysed by SDS-PAGE and Western blotting with sera of sensitized patients and with antiparvalbumin. Protein bands were microsequenced, RNA transcripts were amplified by reverse transcription and polymerase chain reaction (RT-PCR) using primer combinations overlapping the open reading frame. RESULTS: Four IgE and antiparvalbumin reactive proteins(12.5, 24, 38 and 51 kDa) were detected in gel filtration eluate. The cDNA deduced sequence of the 24 kDa protein had 109 amino acid residues with a molecular weight of 11.5 kDa and a theoretical pI of 4.34. The 24 kDa band corresponded therefore to a dimer of a beta-parvalbumin. Its homology was higher with Sal sI than with Gad cI. This new **allergen** was named Gad mI. CONCLUSION: We have characterized a new parvalbumin **allergen** in Gadus morhua. This protein formed oligomers in native and in **reducing** conditions. Gad mI and Gad cI may correspond to two distinct genes of Gadus species.

L8 ANSWER 27 OF 59 MEDLINE
AN 2002290480 IN-PROCESS
DN 22026009 PubMed ID: 12030423
TI High magnesium concentration in vitro decreases human leukocyte activation.
AU Bussiere F I; Mazur A; Fauquert J L; Labbe A; Rayssiguier Y; Tridon A
CS Centre de Recherches en Nutrition Humaine, Unite Maladies Metaboliques et Micronutriments, INRA-Theix, Saint-Genes-Champanelle, France.
SO MAGNESIUM RESEARCH, (2002 Mar) 15 (1-2) 43-8.
Journal code: 8900948. ISSN: 0953-1424.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS IN-PROCESS; NONINDEXED; Priority Journals
ED Entered STN: 20020528
Last Updated on STN: 20020528
AB In view of experimental data suggesting that pharmacological magnesium (Mg) therapy could be expected to temper hypersensitivity, the aim of the present study was to assess the effect of in vitro high Mg concentration (8 mmol/l vs. 0.8 mmol/l) on human leukocyte activation. The first experiment in nine healthy volunteers was performed on total leukocyte suspension containing 82 +/- 4 per cent of neutrophils. The results demonstrate the inhibitory effect of high Mg concentration as shown by the significant reduction of superoxide anion production following phorbol myristate acetate (PMA) or formyl-methionyl-leucyl-phenylalanine (fMLP) activation. Moreover, neutrophils activated with fMLP showed an increased respiratory burst when incubated in low Mg concentration (0.2 mmol/l) as compared to normal Mg concentration (0.8 mmol/l). Similarly, high concentration of Mg resulted in a significant **reduction** in superoxide anion production by eosinophils in response to PMA in five eosinophilic patients. In patients showing Hymenoptera venom hypersensitivity, high Mg concentration resulted in a significant **reduction** of sulphidoleukotrienes production by leukocytes in response to venom **allergen** (six patients) or in response to zymosan activated particules (fourteen patients). Taken together, the

results suggests that Mg acts via a non specific mechanism and appears to be non specific to a particular cell type. As Mg counteracts **calcium** in many physiological and pathological processes, it is reasonable to hypothesise that extracellular Mg can diminish leukocyte activation by its calcium antagonism.

L8 ANSWER 28 OF 59 MEDLINE
AN 2002000351 MEDLINE
DN 21590492 PubMed ID: 11733571
TI Essential role of the prosurvival bcl-2 homologue A1 in mast cell survival after **allergic** activation.
AU Xiang Z; Ahmed A A; Moller C; Nakayama K; Hatakeyama S; Nilsson G
CS Research Group on Mast Cell Biology, Department of Genetics and Pathology, Rudbeck Laboratory, Uppsala University, 751 85 Uppsala, Sweden.
SO JOURNAL OF EXPERIMENTAL MEDICINE, (2001 Dec 3) 194 (11) 1561-69. Journal code: 2985109R. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200201
ED Entered STN: 20020102
Last Updated on STN: 20020125
Entered Medline: 20020111
TI Essential role of the prosurvival bcl-2 homologue A1 in mast cell survival after **allergic** activation.
AB Mast cells reside in tissues, where upon activation through the high-affinity-IgE-receptor (FcepsilonRI) they degranulate and orchestrate the **allergic** reaction. Mast cells survive this activation and can thus be reactivated. In this study we demonstrate that this process depends on the pro-survival gene A1. Activation of mast cells through FcepsilonRI resulted in degranulation, strong induction of A1 mRNA and protein, and cell survival. In contrast, A1-deficient mast cells released granule mediators similar to the wild-type **control**, but the cells did not survive an **allergic** activation. Furthermore, A1(-/-) mice that had been sensitized and provoked with **allergen** exhibited a lower number of mast cell compared with littermate **controls**. The induction of A1 was dependent on **calcium**, as EDTA prevented A1 expression. The **calcium** ionophore, ionomycin, induced A1 expression and mast cell survival, whereas compound 48/80, a well-known mast cell secretagogue, did not. This study uncovers the importance of A1 for mast cell survival in **allergic** reactions, and it proposes A1 as a potential target for the treatment of **allergic** diseases.

L8 ANSWER 29 OF 59 MEDLINE
AN 2001227658 MEDLINE
DN 21135823 PubMed ID: 11240950
TI Enhanced production of leukotrienes by peripheral leukocytes and specific IgE antibodies in patients with chronic obstructive pulmonary disease.
AU Mitsunobu F; Mifune T; Hosaki Y; Ashida K; Tsugeno H; Okamoto M; Takata S; Tanizaki Y
CS Department of Medicine, Misasa Medical Branch, Okayama University Medical School, Japan.
SO JOURNAL OF ALLERGY AND CLINICAL IMMUNOLOGY, (2001 Mar) 107 (3) 492-8. Journal code: 1275002. ISSN: 0091-6749.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200104
ED Entered STN: 20010502
Last Updated on STN: 20010502

Entered Medline: 20010426

AB BACKGROUND: How leukotrienes (LTs) and IgE-mediated **allergy** reflect clinical features in patients with chronic obstructive pulmonary disease (COPD) remains unclear. OBJECTIVE: Our goal was to determine whether LTB4 and LTC4 would correlate with airway obstruction and whether IgE-mediated **allergy** would influence the generation of LTs and bronchial hyperresponsiveness in patients with COPD. METHODS: We measured the pulmonary function, methacholine bronchial hyperresponsiveness, and generation of LTB4 and LTC4 from peripheral leukocytes stimulated with **calcium** ionophore A23187 in relation to the presence of specific IgE antibodies against inhalant **allergens**. RESULTS: The leukocytes of patients with COPD generated significantly more LTB4 (with **allergy**, P <.001; without **allergy**, P <.001) and LTC4 (with **allergy**, P <.001; without **allergy**, P <.01) than the leukocytes of the **control** subjects. LTC4 production was significantly higher in the **allergic** COPD subjects than in the nonallergic COPD patients (P <.01), but the amount of LTB4 generated was not significantly different. FEV(1) significantly correlated with the level of both LTB4 (with **allergy**, r = -0.556, P =.0375; without **allergy**, r = -0.731, P =.0046) and LTC4 (with **allergy**, r = -0.764, P =.0043; without **allergy**, r = -0.526, P =.0414) generation in COPD. The log(10) of the minimum dose of methacholine was significantly higher in COPD patients without **allergy** than in those with **allergy** (P <.05). CONCLUSION: Enhanced LT generation from peripheral leukocytes is observed in patients with COPD, and the presence of specific IgE antibodies against inhalant **allergens** enhances LTC4 generation, bronchial hyperresponsiveness, and the relationship between LTC4 generation and airway obstruction.

L8 ANSWER 30 OF 59 MEDLINE
AN 2001209854 MEDLINE
DN 21194558 PubMed ID: 11298006
TI **Allergens** from fish and egg.
AU Poulsen L K; Hansen T K; Norgaard A; Vestergaard H; Stahl Skov P; Bindslev-Jensen C
CS Allergy Unit, National University Hospital, Copenhagen, Denmark.. lkpallgy@inet.uni2.dk
SO ALLERGY, (2001) 56 Suppl 67 39-42. Ref: 21
Journal code: 7804028. ISSN: 0105-4538.
CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200108
ED Entered STN: 20010820
Last Updated on STN: 20010820
Entered Medline: 20010816

TI **Allergens** from fish and egg.

AB **Allergens** from fish and egg belong to some of the most frequent causes of food **allergic** reactions reported in the literature. Egg **allergens** have been described in both white and yolk, and the egg white proteins ovomucoid, ovalbumin, ovotransferrin and lysozyme have been adopted in the **allergen** nomenclature as Gal d1-d4. The most reported **allergen** from egg yolk seems to be alpha-livitin. In fish, the dominating **allergen** is the homologues of Gad c1 from cod, formerly described as protein M. A close cross-reactivity exists within different species of fish between this **calcium**-binding protein family, denominated the parvalbumins. This cross-reactivity has been indicated to be of clinical relevance for several species, since patients with a positive double-blind, placebo-**controlled** food challenge to cod will also react with other fish species, such as herring,

plaice and mackerel. In spite of the importance of these two **allergen** systems, only a few studies have been performed, and the scarcity of cloned **allergens** from both of the systems is emphasized.

CT Check Tags: Animal; Human
Allergens: CL, classification
***Allergens: IM, immunology**
Chickens
Egg Proteins: AE, adverse effects
*Egg Proteins: IM, immunology
*Fish Products: AE, adverse effects
Fishes: IM, immunology
*Food Hypersensitivity: IM, immunology

CN 0 (**Allergens**); 0 (Egg Proteins)

L8 ANSWER 31 OF 59 MEDLINE
AN 2000410489 MEDLINE
DN 20401256 PubMed ID: 10944959
TI [The role of heparin in **allergic** inflammation].
Udzial heparyny w zapaleniu alergicznym.
AU Jerzynska J; Stelmach I; Kuna P
CS Oddzialu Interny Dzieciecej i Alergologii Wojewodzkiego Szpitala Specjalistycznego w Zgierzu.
SO POLSKI MERKURIUSZ LEKARSKI, (2000 May) 8 (47) 341-6. Ref: 52
Journal code: 9705469. ISSN: 1426-9686.
CY Poland
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA Polish
FS Priority Journals
EM 200008
ED Entered STN: 20000907
Last Updated on STN: 20000907
Entered Medline: 20000831
TI [The role of heparin in **allergic** inflammation].
Udzial heparyny w zapaleniu alergicznym.
AB Heparin is a glycosaminoglican used in prophylactic and treatment of thrombosis. Heparin possesses also non-anticoagulant properties, including modulation of various proteases, anticomplement activity, and anti-inflammatory actions. Inhaled heparin has been shown to **reduce** early phase of asthmatic reaction and suppress **allergen** induced rise in bronchial hyperreactivity. Heparin inhibits the acute cutaneous reaction due to **allergens**. Moreover, inhaled heparin prevents exercise-induced asthma. The exact mechanism of heparin action in bronchial asthma remains obscure. It has been observed that heparin acts as a specific blocker of IP3 receptors and inhibits IP3-mediated **calcium** release in various cell types, including vascular smooth muscle and airway smooth muscle. In this mechanism heparin inhibits **allergen** induced mast cell degranulation and prevents subsequent development of reaction cascade leading to inflammation, bronchial hyperreactivity and asthma. It also modulates migration of proinflammatory cells, eosinophils and neutrophils, into the site of **allergic** reaction. Furthermore, heparin inhibits the increased vascular permeability induced by a wide range of agonists acting via specific receptors located on the vascular endothelial cells. The cationic peroxidases, such as major basic protein and eosinophil peroxidase, are neutralized by the highly anionic heparin; thus heparin inhibits the epithelial damage induced by some of these cationic proteins. The mechanism involved in the control of bronchial hyperreactivity by heparin has been studied little and is yet poorly understood. Heparin deserves further investigations in large number of subjects to provide further insight into the pathophysiology of asthma.

Heparin may also be of clinical importance and may form the basis of novel therapeutic approaches.

L8 ANSWER 32 OF 59 MEDLINE
AN 2000268281 MEDLINE
DN 20268281 PubMed ID: 10764710
TI Purification, biochemical, and immunological characterisation of a major food **allergen**: different immunoglobulin E recognition of the apo- and **calcium**-bound forms of carp parvalbumin.
AU Bugajska-Schretter A; Grote M; Vangelista L; Valent P; Sperr W R; Rumpold H; Pastore A; Reichelt R; Valenta R; Spitzauer S
CS Institute of Medical and Chemical Laboratory Diagnostics, AKH, University of Vienna, Austria.
SO GUT, (2000 May) 46 (5) 661-9.
Journal code: 2985108R. ISSN: 0017-5749.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200006
ED Entered STN: 20000613
Last Updated on STN: 20000613
Entered Medline: 20000601
TI Purification, biochemical, and immunological characterisation of a major food **allergen**: different immunoglobulin E recognition of the apo- and **calcium**-bound forms of carp parvalbumin.
AB BACKGROUND: Almost 4% of the population suffer from food **allergy** which is an adverse reaction to food with an underlying immunological mechanism. AIMS: To characterise one of the most frequent IgE defined food **allergens**, fish parvalbumin. METHODS: Tissue and subcellular distribution of carp parvalbumin was analysed by immunogold electron microscopy and cell fractionation. Parvalbumin was purified to homogeneity, analysed by mass spectrometry and circular dichroism (CD) spectroscopy, and its **allergenic** activity was analysed by IgE binding and basophil histamine release tests. RESULTS: The isoelectric point (pI) 4.7 form of carp parvalbumin, a three EF-hand **calcium** -binding protein, was purified to homogeneity. CD analysis revealed a remarkable stability and refolding capacity of **calcium**-bound parvalbumin. This may explain why parvalbumin, despite cooking and exposure to the gastrointestinal tract, can sensitise patients. Purified parvalbumin reacted with IgE of more than 95% of individuals **allergic** to fish, induced dose-dependent basophil histamine release and contained, on average, 83% of the IgE epitopes present in other fish species. **Calcium** depletion **reduced** the IgE binding capacity of parvalbumin which, according to CD analysis, may be due to conformation-dependent IgE recognition. CONCLUSIONS: Purified carp parvalbumin represents an important cross reactive food **allergen** . It can be used for in vitro and in vivo diagnosis of fish-induced food **allergy**. Our finding that the apo-form of parvalbumin had a greatly **reduced** IgE binding capacity indicates that this form may be a candidate for safe immunotherapy of fish-related food **allergy**.
CT Check Tags: Animal; Human; Support, Non-U.S. Gov't
Allergens: IM, immunology
*Carps: IM, immunology
Cell Fractionation
Circular Dichroism
*Food Hypersensitivity: IM, immunology
Histamine Release: IM, immunology
Immunoblotting
*Immunoglobulin E: IM, immunology
Microscopy, Electron
Parvalbumins: AE, adverse effects

*Parvalbumins: IM, immunology
Parvalbumins: IP, isolation & purification
Spectrum Analysis, Mass
CN 0 (**Allergens**); 0 (Parvalbumins)

L8 ANSWER 33 OF 59 MEDLINE
AN 2000214597 MEDLINE
DN 20214597 PubMed ID: 10752923
TI Effect of misoprostol on the secretion of histamine from basophils of whole blood.
AU Babakhin A A; Nolte H; DuBuske L M
CS Laboratory of Control and Regulation of Allergic Response, Institute of Immunology, Moscow, Russia.
SO ANNALS OF ALLERGY, ASTHMA, AND IMMUNOLOGY, (2000 Mar) 84 (3) 361-5.
Journal code: 9503580. ISSN: 1081-1206.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200004
ED Entered STN: 20000505
Last Updated on STN: 20000505
Entered Medline: 20000427
AB BACKGROUND: Misoprostol (MSP), the synthetic prostaglandin E1 (PGE1) analog, possesses multifunctional features, including modulating some inflammatory aspects of immune and **allergic** disorders. OBJECTIVES: To investigate the effect of MSP on histamine release (HR) from basophils of whole blood using anti-IgE, specific **allergens**, and **calcium** ionophore. METHODS: The study was performed using the automated glass fiber-based whole blood leukocyte histamine release test (LHRT). RESULTS: Very low concentrations of MSP produced a marked inhibition of HR induced with anti-IgE. Maximum inhibition was observed at 10-9 M. It was also shown that the levels of HR inhibition with MSP varied at different incubation times. The greatest inhibition of HR was noted at 1 to 2 hours of incubation at MSP concentrations of 10-8 and 10-9 M, respectively. Incubation of blood from **allergic** patients at the optimal MSP concentration and optimal elapsed time (2 hours) resulted in significant **reductions** of **allergen**-specific HR induced by both Timothy **pollen** grass **allergen** and D.pteronissinus. Incubation of blood with varying concentrations of MSP and subsequent stimulation with **calcium** ionophore A23187 also inhibited HR from basophils. In the latter case, the most effective concentrations of MSP ranged from 10-8 to 10-6 M. CONCLUSIONS: This study demonstrated that MSP can inhibit basophil HR indicating a potentially beneficial role of PGE1 analogs as pharmacotherapy for **allergic** diseases.

L8 ANSWER 34 OF 59 MEDLINE
AN 1999401124 MEDLINE
DN 99401124 PubMed ID: 10469821
TI Keeping children with exercise-induced asthma active.
AU Milgrom H; Taussig L M
CS Department of Pediatrics, National Jewish Medical and Research Center and the University of Colorado Health Sciences Center, Denver, Colorado 80206, USA.. milgromh@njc.org
SO PEDIATRICS, (1999 Sep) 104 (3) e38. Ref: 55
Journal code: 0376422. ISSN: 1098-4275.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals

EM 199909
ED Entered STN: 19991005
Last Updated on STN: 20010521
Entered Medline: 19990917

AB Exercise-induced bronchospasm, exercise-induced bronchoconstriction, and exercise-induced asthma (EIA) are all terms used to describe the phenomenon of transient airflow obstruction associated with physical exertion. It is a prominent finding in children and young adults because of their greater participation in vigorous activities. The symptoms shortness of breath, cough, chest tightness, and wheezing normally follow the brief period of bronchodilation present early in the course of exercise. Bronchospasm typically arises within 10 to 15 minutes of beginning exercise, peaks 8 to 15 minutes after the exertion is concluded, and resolves about 60 minutes later, but it also may appear during sustained exertion. EIA occurs in up to 90% of asthmatics and 40% of patients with **allergic** rhinitis; among athletes and in the general population its prevalence is between 6% and 13%. EIA frequently goes undiagnosed. Approximately 9% of individuals with EIA have no history of asthma or **allergy**. Fifty percent of children with asthma who gave a negative history for EIA had a positive response to exercise challenge.6 Among high school athletes, 12% of subjects not considered to be at risk by history or baseline spirometry tested positive. Before the 1984 Olympic games, of 597 members of the US team, 67 (11%) were found to have EIA. Remarkably, only 26 had been previously identified, emphasizing the importance of screening for EIA even in well-conditioned individuals who appear to be in excellent health. The severity of bronchospasm in EIA is related to the level of ventilation, to heat and water loss from the respiratory tree, and also to the rate of airway rewarming and rehydration after the challenge. Postexercise decrease in the peak expiratory flow rate of normal children may be as much as 15%; therefore, only a decrease in excess of 15% should be viewed as diagnostic. EIA is usually provoked by a workload sufficient to produce 80% of maximum oxygen consumption; however, in severe asthmatics even minimal exertion may be enough to produce symptoms. Patients with normal lung function at rest may have severe air flow limitation induced by exercise,10 and as many as 50% of patients who are well-**controlled** with inhaled corticosteroids still exhibit EIA. A challenge of sufficient magnitude will provoke EIA in all patients with asthma. PHARMACOLOGIC THERAPY: Exercise, unlike exposure to **allergens**, does not produce a long-term increase in airway reactivity. Accordingly, patients whose symptoms manifest only after strenuous activity may be treated prophylactically and do not require continuous therapy. Most asthma medications, even some unconventional ones such as heparin, furosemide, **calcium** channel blockers, and terfenadine, given before exercise, suppress EIA. McFadden accounts for the efficacy of these disparate classes of drugs by their potential effect on the bronchial vasculature that modulates the cooling and/or rewarming phases of the reaction. Short-acting -agonists provide protection in 80% to 95% of affected individuals with insignificant side effects and have been regarded for many years as first-line therapy. Two long-acting bronchodilators, salmeterol and formoterol, have been found effective in the prevention of EIA.18-21 A single 50-microg dose of salmeterol protects against EIA for 9 hours; its duration appears to wane in the course of daily therapy. Cromolyn sodium is highly effective in 70% to 87% of those diagnosed with EIA and has minimal side effects. Nedocromil sodium provides protection equal to that of cromolyn in children. Children commonly engage in unplanned physical activity and sometimes are not allowed to carry their own medication. Thus, a simple long-acting regimen given at home is likely to be more effective than short-acting drugs that must be administered in a timely manner. Although the 12-hour protection by salmeterol reported by Bronsky et al may not persist with continued use, the 9-hour duration of action is

L8 ANSWER 35 OF 59 MEDLINE

AN 1999002973 MEDLINE
DN 99002973 PubMed ID: 9784653
TI Identification of **allergens** in oilseed rape (Brassica napus) pollen.
AU Focke M; Hemmer W; Hayek B; Gotz M; Jarisch R
CS Dermatologic and Pediatric Allergy Clinic, Wilhelminen Hospital, Vienna, Austria.
SO INTERNATIONAL ARCHIVES OF ALLERGY AND IMMUNOLOGY, (1998 Oct) 117 (2) 105-12.
Journal code: 9211652. ISSN: 1018-2438.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199811
ED Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981116
TI Identification of **allergens** in oilseed rape (Brassica napus) pollen.
AB BACKGROUND: Pollen from oilseed rape (OSR), Brassica napus, an increasingly cultivated oilplant from the Brassicaceae, has been recognized as a potential cause of **allergic** sensitization. **Allergens** have been hardly investigated. METHODS: We characterized IgE binding proteins in OSR pollen by immunoblot, immunoblot inhibition and specific monoclonal antibodies using sera from 89 patients sensitized to OSR. RESULTS: Two low-molecular-weight **allergens** of 6/8 kD and 14 kD as well as a high molecular-weight cluster (27-69 kD) comprising six cross-reactive peptides could be identified. The three **allergens** were recognized by 50, 34 and 80% of patients, respectively. Immunoblot IgE binding to OSR could be totally inhibited by rye **pollen** and moderately by birch **pollen** (6/8 and 14 kD) while mugwort had little effect. An anti-profilin-specific monoclonal antibody bound specifically to a 14-kD protein in OSR. Binding to the 6/8-kD rape **allergen** could be effectively inhibited by rAln g 2, a **calcium**-binding protein from alder. Periodate treatment led to a significant **reduction** in IgE binding to the 27 to 69-kD OSR **allergens** indicating that carbohydrate determinants are involved in IgE binding. OSR proteins were capable to quench IgE binding to timothy grass **pollen** proteins of >/=60 kD suggesting that grass **pollen** group 4 **allergens** cross-react with the 27 to 69-kD cluster in OSR. CONCLUSIONS: The data demonstrate that OSR **pollen** is **allergenic** and indicate that the identified **allergens** represent cross-reacting homologues of well-known **pollen allergens**, i.e. **calcium**-binding proteins, profilins, and high-molecular-weight glycoproteins. Via cross-reactivity, exposure to OSR **pollen** may be a prolonging and aggravating factor in underlying birch and grass **pollen** **allergy**.
CT Check Tags: Animal; Human
***Allergens: IM, immunology**
Antibodies, Monoclonal: IM, immunology
*Brassica: IM, immunology
Cross Reactions: IM, immunology
Electrophoresis, Polyacrylamide Gel
Hypersensitivity, Immediate: IM, immunology
Immunoblotting
Immunoglobulin E: IM, immunology
Immunoglobulin G: IM, immunology
Membrane Glycoproteins: IM, immunology
Mice
Molecular Weight
*Plant Oils

Plant Proteins: IM, immunology
*Pollen: IM, immunology
Rabbits
Skin Tests
CN 0 (**Allergens**); 0 (Antibodies, Monoclonal); 0 (Immunoglobulin G); 0 (Membrane Glycoproteins); 0 (Plant Oils); 0 (Plant Proteins)

L8 ANSWER 36 OF 59 MEDLINE
AN 1998413866 MEDLINE
DN 98413866 PubMed ID: 9742934
TI Engineering of hypoallergenic mutants of the Brassica pollen **allergen**, Bra r 1, for immunotherapy.
AU Okada T; Swoboda I; Bhalla P L; Toriyama K; Singh M B
CS Laboratory of Plant Breeding and Genetics, Graduate School of Agricultural Science, Tohoku University, Sendai, Japan.
SO FEBS LETTERS, (1998 Sep 4) 434 (3) 255-60.
Journal code: 0155157. ISSN: 0014-5793.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199810
ED Entered STN: 19981020
Last Updated on STN: 19981020
Entered Medline: 19981008
TI Engineering of hypoallergenic mutants of the Brassica pollen **allergen**, Bra r 1, for immunotherapy.
AB The Brassica **pollen allergen** Bra r 1 belongs to a new family of Ca2+-binding proteins, characterized by the presence of two potential EF-hand **calcium**-binding domains. Disruption of these EF-hand motifs by amino acid substitutions demonstrated that both domains of Bra r 1 constitute functional Ca2+-binding sites. **Calcium**-binding deficient mutants displayed significantly **reduced** IgE-binding activity. Injection of these mutated Bra r 1 variants into a murine model system showed that mouse IgG raised against the mutants recognized native Bra r 1 in Brassica **pollen** extracts suggesting the potential use of the engineered **allergens** for effective immunotherapy.
CT Check Tags: Animal; Support, Non-U.S. Gov't
***Allergens: GE, genetics**
Allergens: ME, metabolism
Allergens: TU, therapeutic use
Amino Acid Sequence
Base Sequence
*Brassica: IM, immunology
Calcium: ME, metabolism
DNA Primers
Enzyme-Linked Immunosorbent Assay
Immunoglobulin E: BI, biosynthesis
*Immunotherapy
Mice
Molecular Sequence Data
Mutagenesis, Site-Directed
*Pollen: IM, immunology
Protein Binding
Recombinant Proteins: GE, genetics
Recombinant Proteins: ME, metabolism
Recombinant Proteins: TU, therapeutic use
CN 0 (**Allergens**); 0 (DNA Primers); 0 (Recombinant Proteins)

L8 ANSWER 37 OF 59 MEDLINE
AN 1998109557 MEDLINE
DN 98109557 PubMed ID: 9449503

TI Parvalbumin, a cross-reactive fish **allergen**, contains IgE-binding epitopes sensitive to periodate treatment and Ca2+ depletion.
AU Bugajska-Schretter A; Elfman L; Fuchs T; Kapiotis S; Rumpold H; Valenta R; Spitzauer S
CS Institute of Medical and Chemical Laboratory Diagnostics, AKH, University of Vienna, Austria.
SO JOURNAL OF ALLERGY AND CLINICAL IMMUNOLOGY, (1998 Jan) 101 (1 Pt 1) 67-74. Journal code: 1275002. ISSN: 0091-6749.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199802
ED Entered STN: 19980217
Last Updated on STN: 19980217
Entered Medline: 19980205
TI Parvalbumin, a cross-reactive fish **allergen**, contains IgE-binding epitopes sensitive to periodate treatment and Ca2+ depletion.
AB BACKGROUND: Type I **allergy** to fish is a severe health problem in countries in which a large percentage of the population derive income from fishing. OBJECTIVE: The aim of the study was to characterize cross-reactive IgE-binding components in six different fish species (cod, tuna, salmon, perch, carp, and eel). The effect of **reducing** extraction conditions, periodate treatment, and depletion of Ca2+ on binding of IgE to the **allergens** was investigated. METHODS: Extracts were prepared under nonreducing and **reducing** conditions. IgE-binding components were characterized by IgE immunoblotting, and cross-reactive epitopes were studied by IgE-immunoblot inhibition experiments. To reveal **calcium**-sensitive or carbohydrate-containing epitopes, nitrocellulose-blotted extracts were exposed to ethylene glycol bis(beta-aminoethyl ether)-N,N,N',N'-tetraacetic acid (EGTA) and periodate. RESULTS: Sera from all patients **allergic** to fish (n = 30) displayed IgE reactivity to parvalbumin, a 12 kd protein present in fish extracts from six different species. **Reducing** extraction conditions had no effect on IgE binding to parvalbumins, whereas periodate treatment and depletion of protein-bound **calcium** led to a substantial **reduction** of IgE binding. Parvalbumins from six different species contained cross-reactive IgE epitopes. CONCLUSION: Parvalbumin represents a cross-reactive fish **allergen**. It contains IgE epitopes that are sensitive to periodate treatment and Ca2+-depletion.
CT Check Tags: Animal; Comparative Study; Human; Support, Non-U.S. Gov't
***Allergens: CH, chemistry**
Allergens: IP, isolation & purification
Calcium
Cross Reactions
Epitopes: CH, chemistry
Epitopes: IP, isolation & purification
*Fishes: IM, immunology
Food Hypersensitivity: ET, etiology
Food Hypersensitivity: IM, immunology
Immunochemistry
Immunoglobulin E: BL, blood
Parvalbumins: CH, chemistry
*Parvalbumins: IM, immunology
Parvalbumins: IP, isolation & purification
Periodic Acid
Species Specificity
CN 0 (**Allergens**); 0 (Epitopes); 0 (Parvalbumins)

L8 ANSWER 38 OF 59 MEDLINE
AN 1998005106 MEDLINE
DN 98005106 PubMed ID: 9345295

TI Molecular characterization, expression in Escherichia coli, and epitope analysis of a two EF-hand **calcium**-binding birch **pollen allergen**, Bet v 4.
AU Twardosz A; Hayek B; Seiberler S; Vangelista L; Elfman L; Gronlund H; Kraft D; Valenta R
CS Institute of General and Experimental Pathology, AKH, University of Vienna, Austria.
SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1997 Oct 9) 239 (1) 197-204.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-Y12560
EM 199711
ED Entered STN: 19971224
Last Updated on STN: 19990129
Entered Medline: 19971124
TI Molecular characterization, expression in Escherichia coli, and epitope analysis of a two EF-hand **calcium**-binding birch **pollen allergen**, Bet v 4.
AB Birch pollen belongs to the most potent elicitors of Type I **allergic** reactions in early spring. Using serum IgE from a birch **pollen allergic** patient, two cDNA clones (clone 6 and clone 13) were isolated from a birch **pollen** expression cDNA library constructed in phage lambda gt11. Clone 6 encoded a 9.3 kD two EF-hand **calcium**-binding protein, designated Bet v 4, with significant end to end sequence homology to EF-hand **calcium** -binding **allergens** from weed and grass **pollen**. Recombinant Bet v 4, expressed as beta-galactosidase fusion protein, reacted with serum IgE from approximately 20% of **pollen allergic** individuals. Depletion of **allergenbound calcium** by EGTA treatment lead to a substantial **reduction** of IgE-binding to Bet v 4, indicating that protein-bound **calcium** is necessary for the maintenance of IgE-epitopes. The greatly **reduced** IgE-binding capacity of clone 13, a Bet v 4 fragment that lacked the 16 N-terminal amino acids, indicated that the N-terminus contributes significantly to the proteins IgE-binding capacity. By IgE-inhibition experiments it was demonstrated that recombinant Bet v 4 shared IgE-epitopes with natural Bet v 4 and a homologous timothy grass pollen **allergen**. Recombinant Bet v 4 may therefore be considered as a relevant crossreactive plant **allergen**, which may be used for diagnosis and treatment of patients suffering from multivalent plant **allergies**.
CT Check Tags: Comparative Study; Support, Non-U.S. Gov't
***Allergens: GE, genetics**
Allergens: IP, isolation & purification
Allergens: ME, metabolism
Amino Acid Sequence
Antibodies, Anti-Idiotypic: ME, metabolism
Base Sequence
Brassica
*Calcium-Binding Proteins: GE, genetics
Calcium-Binding Proteins: IP, isolation & purification
Calcium-Binding Proteins: ME, metabolism
Cloning, Molecular
DNA, Plant: CH, chemistry
Epitopes, B-Lymphocyte: AN, analysis
Escherichia coli
Molecular Sequence Data
*Plant Proteins: GE, genetics
Plant Proteins: IP, isolation & purification

Plant Proteins: ME, metabolism
Poaceae
Protein Binding
Recombinant Proteins: ME, metabolism
Sequence Alignment
Trees

CN 0 (**Allergens**); 0 (Antibodies, Anti-Idiotypic); 0 (Bet v 4 **allergen**); 0 (Calcium-Binding Proteins); 0 (DNA, Plant); 0 (Epitopes, B-Lymphocyte); 0 (Plant Proteins); 0 (Recombinant Proteins); 0 (anti-IgE)

L8 ANSWER 39 OF 59 MEDLINE
AN 97170258 MEDLINE
DN 97170258 PubMed ID: 9017787
TI Use of **allergen** bronchoprovocation to screen drugs for anti-asthma activity.
AU Hendeles L; Harman E
CS Department of Pharmacy Practice, College of Pharmacy, University of Florida, Gainesville, USA.. Hendeles@COP.HEALTH.UFL.EDU
NC RR00082 (NCRR)
SO PHARMACOTHERAPY, (1997 Jan-Feb) 17 (1 Pt 2) 39S-49S. Ref: 53
Journal code: 8111305. ISSN: 0277-0008.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199704
ED Entered STN: 19970422
Last Updated on STN: 19970422
Entered Medline: 19970408
TI Use of **allergen** bronchoprovocation to screen drugs for anti-asthma activity.
AB In the atopic patient with asthma, **allergens** are an important cause of chronic airway inflammation and symptoms. Natural exposure to seasonal **allergens**, such as grass **pollen**, may result in exacerbation of asthma, increased airway responsiveness (i.e., increased susceptibility of the airways to constrict), and an increased frequency of emergency room visits. Removal of patients from exposure to indoor **allergens**, such as dust **mites**, results in a marked **reduction** in symptoms, less airway responsiveness, and a decrease in drug requirements. In the pulmonary function laboratory, inhalation of increasing doses of **allergen**, in a safe and **controlled** manner (**allergen** bronchoprovocation), produces physiological responses similar to those observed after natural exposure. These include an immediate decrease in the forced expiratory volume in 1 second (FEV1) that is rapid in onset but short in duration (early response), a subsequent gradual decline in FEV1 4-8 hours after **allergen** inhalation that is sustained (late response), an increase in airway responsiveness, and infiltration of the airway mucosa by inflammatory cells. Drugs that are effective as maintenance therapy for chronic asthma generally attenuate the late response to **allergen** bronchoprovocation, and those with antiinflammatory effects (e.g., inhaled corticosteroids) also attenuate the **allergen**-induced increase in airway responsiveness and cellular infiltration of the airways. However, the magnitude of drug effect in this clinical model does not correlate well with the drug's relative efficacy in chronic asthma. In contrast, drugs that have no effect in this clinical model, such as **calcium** channel blockers, ketotifen, and antihistamines, are ineffective as maintenance therapy for chronic asthma. Thus, it appears that **allergen** bronchoprovocation is most useful as a screening tool for excluding drugs that are unlikely to be effective for chronic asthma and

for determining whether a drug has antiinflammatory and/or immunomodulatory actions on the airway mucosa.
CT Check Tags: Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Allergens: IM, immunology
*Anti-Asthmatic Agents: TU, therapeutic use
*Asthma: DT, drug therapy
Asthma: PP, physiopathology
Bronchial Provocation Tests
Drug Evaluation
CN 0 (**Allergens**); 0 (Anti-Asthmatic Agents)

L8 ANSWER 40 OF 59 MEDLINE
AN 97024471 MEDLINE
DN 97024471 PubMed ID: 8870699
TI Diminished interferon-gamma (IFN-gamma) production by bacterial antigen-specific T cells in atopic patients.
AU Shimojo N; Kohno Y; Katsuki T; Hoshioka A; Honma K; Saito K; Niimi H
CS Department of Paediatrics, Chiba University School of Medicine, Japan.
SO CLINICAL AND EXPERIMENTAL IMMUNOLOGY, (1996 Oct) 106 (1) 62-6.
Journal code: 0057202. ISSN: 0009-9104.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199612
ED Entered STN: 19970128
Last Updated on STN: 19970128
Entered Medline: 19961210
AB In this study, we established and studied cytokine production of T cell lines (TCL) specific to either a purified protein derivative of Mycobacterium tuberculosis (PPD) or Dermatophagoides farinae (Df) from atopic patients and non-atopic healthy subjects. IFN-gamma was detected in the culture supernatants of all of 36 PPD-specific TCL established from healthy controls, whereas only 24 of 38 PPD-specific TCL from patients produced IFN-gamma. Furthermore, the amounts of IFN-gamma produced by PPD-specific TCL from patients were significantly lower than those from healthy controls. No IL-4 was detected in any PPD-specific TCL from either healthy controls or atopic patients. The amounts of IL-4 production from Df-specific TCL from atopic patients were much higher than from healthy **controls**, while few TCL produced IFN-gamma. These results suggest that the skewing to the Th2-type T cell response in atopic patients is a response not only to **allergens**, but also to bacterial antigens, compared with non-atopic subjects. Activation of PPD-specific TCL from patients with **calcium** ionophore A23187 plus phorbol myristate acetate resulted in much higher IFN-gamma production than in TCL established from healthy **controls**, indicating that the low production of IFN-gamma by PPD-specific T cells from atopic patients is not due to an intrinsic T cell defect but to some regulatory mechanisms.
CN 0 (Cytokines); 0 (Dermatophagoides **allergens**); 0 (Epitopes); 0 (Glycoproteins); 0 (Tuberculin)

L8 ANSWER 41 OF 59 MEDLINE
AN 96133474 MEDLINE
DN 96133474 PubMed ID: 8543752
TI Seasonal variations of interleukin-4 and interferon-gamma release by peripheral blood mononuclear cells from atopic subjects stimulated by polyclonal activators.
AU Lagier B; Pons N; Rivier A; Chanal I; Chanez P; Bousquet J; Pene J
CS Institut National de Sante et de la Recherche Medicale, Centre Hospitalo-Universitaire, Montpellier, France.
SO JOURNAL OF ALLERGY AND CLINICAL IMMUNOLOGY, (1995 Dec) 96 (6 Pt 1) 932-40.
Journal code: 1275002. ISSN: 0091-6749.
CY United States

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199602
ED Entered STN: 19960227
Last Updated on STN: 19960227
Entered Medline: 19960213
AB IgE synthesis is **controlled** by interleukin (IL)-4 and interferon (IFN)-gamma, but there is heterogeneity in the IL-4 response depending on the sensitization of patients and natural **allergen** exposure. In patients sensitized to various **allergens**, we studied the synthesis of IL-4, IFN-gamma, and IgE to determine to what extent their in vitro immune response may be influenced by **pollen** season, depending on their sensitization. We studied 12 nonallergic individuals, seven patients sensitized to cypress **pollen**, 12 sensitized to grass **pollen**, 14 sensitized to several **pollens**, and 42 patients with polysensitization. The release of IL-4 and IFN-gamma from peripheral blood mononuclear cells stimulated by polyclonal agents (**calcium** ionophore A23187 and phorbol myristate acetate) was measured by ELISA. The spontaneous and IL-4-induced release of IgE was measured by ELISA. In patients with cypress **pollen** **allergy**, IL-4 and IgE release were significantly lower than in patients with other **allergies**. In the **pollen** -sensitized group, IL-4 and IgE release were significantly higher during the pollen season than out of it. No variation in IL-4 or IgE release was observed in the polysensitized group. IFN-gamma production was not affected by the pollen season. These data show that the seasonal variations of IL-4 and IgE synthesis differ according to the sensitization of patients.

L8 ANSWER 42 OF 59 MEDLINE
AN 95122930 MEDLINE
DN 95122930 PubMed ID: 7822663
TI The effect of MK-0591, a novel 5-lipoxygenase activating protein inhibitor, on leukotriene biosynthesis and **allergen**-induced airway responses in asthmatic subjects in vivo.
AU Diamant Z; Timmers M C; van der Veen H; Friedman B S; De Smet M; Depre M; Hilliard D; Bel E H; Sterk P J
CS Department of Pulmonology, University Hospital, Leiden, The Netherlands.
SO JOURNAL OF ALLERGY AND CLINICAL IMMUNOLOGY, (1995 Jan) 95 (1 Pt 1) 42-51.
Journal code: 1275002. ISSN: 0091-6749.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199502
ED Entered STN: 19950223
Last Updated on STN: 19970203
Entered Medline: 19950216
TI The effect of MK-0591, a novel 5-lipoxygenase activating protein inhibitor, on leukotriene biosynthesis and **allergen**-induced airway responses in asthmatic subjects in vivo.
AB BACKGROUND: The 5-lipoxygenase metabolites of arachidonic acid are likely to be involved in the pathophysiology of atopic asthma. We investigated the effect of pretreatment with MK-0591, a novel 5-lipoxygenase activating protein inhibitor, on **allergen**-induced early asthmatic reactions (EARs) and late asthmatic reactions (LARs), and subsequent airway hyperresponsiveness to histamine. METHODS: Eight atopic men with mild to moderate asthma aged 19 to 31 years, (forced expiratory volume in 1 second [FEV1] > or = 67% of predicted value, histamine provocative concentration causing a 20% fall in FEV1 [PC20] < 4 mg/ml) and documented EAR and LAR to

house dust **mite** extract participated in a two-period, double-blind, placebo-**controlled**, crossover study. During each study period histamine PC20 was measured 2 days before and 1 day after a standardized **allergen** inhalation challenge test. MK-0591 was administered in 3 oral doses of 250 mg each at 24, 12, and 1.5 hours before inhalation of **allergen**. Biochemical activity of MK-0591 was determined by **calcium** ionophore A-23187-stimulated leukotriene (LT)B4 biosynthesis in whole blood ex vivo and by urinary LTE4 excretion. Airway response to **allergen** was measured by FEV1 (percent fall from baseline). The EAR (0 to 3 hours) and the LAR (3 to 8 hours) were expressed as corresponding areas under the time-response curves. RESULTS: MK-0591 and placebo did not differ in their effects on prechallenge FEV1 ($p = 0.10$). As compared with the value before pretreatment, MK-0591 blocked LTB4 biosynthesis and LTE4 excretion by a mean of 98% (range, 96% to 99%; $p < 0.002$) and 87% (range, 84% to 96%; $p < 0.046$), respectively, from 0 to 24 hours after **allergen** challenge. Both the EAR and the LAR were significantly **reduced** after administration of MK-0591 as compared with placebo, with a mean inhibition of 79% ($p = 0.011$) and 39% ($p = 0.040$), respectively. **Allergen**-induced airway hyperresponsiveness was not significantly different between the two pretreatment periods ($p = 0.37$). CONCLUSIONS: In this study oral MK-0591 prevented leukotriene biosynthesis after **allergen** challenge in patients with mild to moderate asthma. The results of our study indicate that 5-lipoxygenase products play an important role during the EAR, whereas their contribution to the pathophysiology of the LAR seems to be of less importance.

CT Check Tags: Comparative Study; Human; Male
Administration, Oral
Adult
***Allergens: AE, adverse effects**
Allergens: DU, diagnostic use
*Asthma: DT, drug therapy
Asthma: ME, metabolism
Asthma: PP, physiopathology
*Bronchial Hyperreactivity: DT, drug therapy
Bronchial Hyperreactivity: ME, metabolism
Bronchial Hyperreactivity: PP, physiopathology
Bronchial Provocation Tests: MT, methods
*Carrier Proteins: AI, antagonists & inhibitors
Creatinine: UR, urine
Double-Blind Method
Forced Expiratory Volume: DE, drug effects
Histamine: DU, diagnostic use
*Indoles: AD, administration & dosage
Indoles: BL, blood
*Leukotriene Antagonists
Leukotrienes: AN, analysis
*Leukotrienes: BI, biosynthesis
*Membrane Proteins: AI, antagonists & inhibitors
*Quinolines: AD, administration & dosage
Quinolines: BL, blood
Time Factors

CN 0 (5-lipoxygenase-activating protein); 0 (**Allergens**); 0 (Carrier Proteins); 0 (Indoles); 0 (Leukotriene Antagonists); 0 (Leukotrienes); 0 (Membrane Proteins); 0 (Quinolines)

L8 ANSWER 43 OF 59 MEDLINE
AN 94378107 MEDLINE
DN 94378107 PubMed ID: 8091317
TI Effect of the 5-lipoxygenase inhibitor ZD2138 on **allergen**-induced early and late asthmatic responses.
AU Nasser S M; Bell G S; Hawksworth R J; Spruce K E; MacMillan R; Williams A J; Lee T H; Arm J P

CS Department of Allergy and Allied Respiratory Disorders, UMDS, Guy's Hospital, London.
SO THORAX, (1994 Aug) 49 (8) 743-8.
Journal code: 0417353. ISSN: 0040-6376.
CY ENGLAND: United Kingdom
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Priority Journals
EM 199410
ED Entered STN: 19941031
Last Updated on STN: 19970203
Entered Medline: 19941017
TI Effect of the 5-lipoxygenase inhibitor ZD2138 on **allergen** -induced early and late asthmatic responses.
AB BACKGROUND--Leukotrienes are lipid mediators generated from arachidonic acid by the 5-lipoxygenase pathway which may play an important part in the pathophysiology of asthma. Previous studies have demonstrated attenuation of the **allergen**-induced early and late asthmatic responses by leukotriene receptor antagonists. The effect of the 5-lipoxygenase inhibitor ZD2138, a non-redox lipoxygenase inhibitor which inhibits leukotriene synthesis for 24 hours after single doses of 350 mg, on **allergen**-induced early and late asthmatic responses has been assessed. METHODS--Eight asthmatic subjects with baseline FEV1 > 70% were studied. On screening, all subjects developed an **allergen** -induced biphasic asthmatic response to grass pollen, cat dander, or house dust mite. ZD2138 (350 mg) or placebo was given on two occasions separated by two weeks in a randomised double blind fashion. **Allergen** inhalation challenge was performed four hours after dosing and FEV1 was measured for eight hours. The inhibitory activity of ZD2138 on the 5-lipoxygenase pathway was assessed by measurements of **calcium** ionophore-stimulated generation of LTB4 in whole blood ex vivo and by analysis of urinary LTE4 levels before administration of drug or placebo and at regular intervals after oral drug dosing and **allergen** challenge. RESULTS--ZD2138 produced no significant bronchodilatation or attenuation of the early or late asthmatic response, although there was 82% inhibition of whole blood generation of LTB4 in response to **calcium** ionophore stimulation and 52% **reduction** in urinary excretion of LTE4. CONCLUSIONS--In asthmatic subjects the 5-lipoxygenase inhibitor ZD2138 did not protect against **allergen** -induced asthmatic responses, despite substantial inhibition of 5-lipoxygenase.

L8 ANSWER 44 OF 59 MEDLINE
AN 94036600 MEDLINE
DN 94036600 PubMed ID: 8221508
TI **Allergic** reactions to fish.
AU O'Neil C; Helbling A A; Lehrer S B
CS Department of Medicine, Tulane Medical Center, New Orleans, LA.
NC 5M01RR05096-03 (NCRR)
SO CLINICAL REVIEWS IN ALLERGY, (1993 Summer) 11 (2) 183-200. Ref: 64
Journal code: 8308524. ISSN: 0731-8235.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199312
ED Entered STN: 19940117
Last Updated on STN: 19940117
Entered Medline: 19931210

TI **Allergic** reactions to fish.
AB A wide variety of fish are known to induce **allergic** reactions following ingestion or inhalation of vapors by sensitized individuals. Although the exact prevalence of fish sensitivity is not known, fish are among the most important food **allergens**; and as consumption of fish increases, rates of sensitization are expected to increase. Diagnosis of fish **allergy** is aided by clinical history, skin prick testing, and in vitro assays; however, double-blind placebo-**controlled** food challenges are the most reliable method to confirm fish **allergy** and to identify putative species. It appears from RAST inhibition and SDS-PAGE/Western blot studies that the current policy of recommending that fish-sensitive individuals avoid all species of fish should be reevaluated. The major **allergen** in codfish (Gad cI) is one of the most extensively studied of all **allergens**. It is a **calcium**-chelating protein, with a mol wt of 12,328 kDa and an isoelectric point of 4.75. As an **allergen**, Gad cI is very stable and its **allergenic** activity appears to be dependent on amino acid sequence. Crossreactivity among some fish species may be the result of common structures within related proteins.
CT Check Tags: Animal; Human; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Allergens: IM, immunology
Amino Acid Sequence
Cross Reactions: IM, immunology
*Fishes
*Food Hypersensitivity
Food Hypersensitivity: DI, diagnosis
Food Hypersensitivity: IM, immunology
Food Hypersensitivity: TH, therapy
Molecular Sequence Data
*Seafood: AE, adverse effects
Species Specificity
CN 0 (**Allergens**)

L8 ANSWER 45 OF 59 MEDLINE
AN 93367118 MEDLINE
DN 93367118 PubMed ID: 8360398
TI Immunologic studies of the mechanisms of occupational asthma caused by western red cedar.
AU Frew A; Chan H; Dryden P; Salari H; Lam S; Chan-Yeung M
CS Department of Medicine, Vancouver General Hospital, University of British Columbia, Canada.
SO JOURNAL OF ALLERGY AND CLINICAL IMMUNOLOGY, (1993 Sep) 92 (3) 466-78. Journal code: 1275002. ISSN: 0091-6749.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199309
ED Entered STN: 19931015
Last Updated on STN: 19931015
Entered Medline: 19930930
AB BACKGROUND: Occupational asthma caused by western red cedar (Thuja plicata) is a common problem in sawmill industries. The objective of this study was to examine the cellular and immunologic mechanisms of western red cedar asthma (WRCA) more closely. METHODS: Bronchial biopsy specimens, bronchoalveolar lavage (BAL) mast cells and peripheral blood basophils from patients with WRCA, patients with atopic asthma, and nonatopic **control** subjects were challenged in vitro with plicatic acid (PA), PA-human serum albumin conjugate (PA-HSA), grass **pollen**, or **calcium** ionophore. RESULTS: PA (100 micrograms/ml) released histamine from the basophils of 9 of 11 patients with WRCA, 1 of 7 patients with atopic asthma, and 2 of 7 normal subjects. PA triggered

histamine release from 10 of 11 bronchial biopsy specimens and 8 of 8 BAL samples from patients with WRCA. Interestingly, PA released histamine from BAL cells and bronchial biopsy specimens from 3 of 7 normal subjects but in none of the patients with atopic asthma. PA-HSA-induced histamine release from basophils and biopsy specimens was confined to patients with WRCA. PA-specific IgE was not detectable in serum from most patients with WRCA, and their serum did not transfer PA sensitivity to human lung fragments or lactate-stripped basophils. After pretreatment with anti-IgE in the absence of **calcium**, basophils from 14 subjects with WRCA still responded to PA (mean 64% to 67% of pretreatment response), whereas responses to grass **pollen** or anti-IgE were abolished.
CONCLUSIONS: This study confirms that PA releases histamine from bronchial mast cells of most patients with WRCA but not from those of patients with atopic asthma. The PA response of some normal subjects suggests that PA may have both specific and nonspecific actions on mast cells and basophils, whereas the serologic studies indicate histamine release in WRCA cannot simply be attributed to PA-specific IgE.

CT Check Tags: Female; Human; Male; Support, Non-U.S. Gov't
Adult
***Allergens: DU, diagnostic use**
Allergens: IM, immunology
Analysis of Variance
*Asthma: IM, immunology
Basophils: IM, immunology
Bronchi: IM, immunology
Bronchial Provocation Tests
Bronchoalveolar Lavage Fluid: IM, immunology
Immunoglobulin E: BL, blood
*Naphthols: DU, diagnostic use
Naphthols: IM, immunology
*Occupational Diseases: IM, immunology
*Wood

CN 0 (**Allergens**); 0 (Naphthols)

L8 ANSWER 46 OF 59 MEDLINE
AN 93221016 MEDLINE
DN 93221016 PubMed ID: 8385430
TI Oral leukotriene inhibitor (MK-886) blocks **allergen**-induced airway responses.
AU Friedman B S; Bel E H; Buntinx A; Tanaka W; Han Y H; Shingo S; Spector R; Sterk P
CS Clinical Pharmacology Department, Merck Research Laboratories, Rahway, NJ 07065.
SO AMERICAN REVIEW OF RESPIRATORY DISEASE, (1993 Apr) 147 (4) 839-44.
Journal code: 0370523. ISSN: 0003-0805.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199304
ED Entered STN: 19930521
Last Updated on STN: 19930521
Entered Medline: 19930430
TI Oral leukotriene inhibitor (MK-886) blocks **allergen**-induced airway responses.
AB To elucidate the role of leukotrienes (LT) in **allergic** asthma in humans the effect of MK-886, an LT biosynthesis inhibitor, was evaluated on antigen-induced early (EAR) and late (LAR) asthmatic reactions and bronchial responsiveness to histamine. Eight atopic men participated in a two-part, double-blind, placebo-**controlled**, crossover trial.
MK-886 was administered in two oral doses of 500 mg and 250 mg, 1 h before

and 2 h after **allergen** inhalation, respectively. Biochemical effects of MK-886 were evaluated by the inhibition of urinary LTE4 excretion and **calcium** ionophore-stimulated LTB4 biosynthesis in whole blood ex vivo. MK-886 significantly inhibited the EAR by 58.4% (AUC0-3 h) and the LAR by 43.6% (AUC3-7 h) when compared with placebo ($p < 0.01$). There was no difference in PC20 histamine 30 h post **allergen** challenge between MK-886 and placebo (0.33 and 0.27 doubling doses, $p > 0.1$). MK-886 inhibited **calcium** ionophore-stimulated LTB4 production in whole blood (54.2 +/- 25.6%) for up to 6 h post **allergen** challenge. LTE4 excretion in urine was inhibited by 51.5% during the EAR by as much as 80% during the LAR. This indicates that LT play a role in **allergen**-induced asthmatic reactions in humans in vivo and that LT synthesis inhibitors such as MK-886 should be further explored for the treatment of asthma.

CT Check Tags: Human; Male
Administration, Oral
Adult
***Allergens**
Asthma: ME, metabolism
*Asthma: PP, physiopathology
*Bronchial Provocation Tests
Double-Blind Method
Drug Evaluation
Histamine: DU, diagnostic use
*Indoles: AD, administration & dosage
*Leukotriene Antagonists
Leukotriene E4
SRS-A: AA, analogs & derivatives
SRS-A: BI, biosynthesis
SRS-A: BL, blood

CN 0 (**Allergens**); 0 (Indoles); 0 (Leukotriene Antagonists); 0 (SRS-A)

L8 ANSWER 47 OF 59 MEDLINE
AN 93171506 MEDLINE
DN 93171506 PubMed ID: 8436778
TI Increased plasma platelet-activating factor in children with acute asthmatic attacks and decreased in vivo and in vitro production of platelet-activating factor after immunotherapy.
AU Hsieh K H; Ng C K
CS Department of Pediatrics, College of Medicine, National Taiwan University, Taipei, Republic of China.
SO JOURNAL OF ALLERGY AND CLINICAL IMMUNOLOGY, (1993 Feb) 91 (2) 650-7. Journal code: 1275002. ISSN: 0091-6749.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199303
ED Entered STN: 19930402
Last Updated on STN: 19930402
Entered Medline: 19930322
AB BACKGROUND: To explore the possible role of platelet-activating factor (PAF) in the pathogenesis of bronchial asthma, circulating PAF and in vitro production of PAF were studied. METHODS: Radioimmunoassay kits were used in 15 children with acute asthmatic attacks, in 25 newly diagnosed asthmatic children, in 25 good and 18 poor responders to immunotherapy, and in 18 healthy controls. RESULTS: The results demonstrated the following: (1) PAF was present in the blood of healthy controls. (2) New patients had much higher circulating PAF than did healthy **controls** ($p < 0.005$), and the circulating PAF decreased after immunotherapy in good ($p < 0.005$) but not in poor responders. (3) The circulating PAF increased up to 20 times that of healthy **controls** during acute asthmatic

attacks. (4) The spontaneous and **allergen**-stimulated secretion of PAF were markedly increased in new patients and decreased to normal after successful immunotherapy ($p < 0.005$). (5) No increased spontaneous and **allergen**-stimulated production of PAF was found during acute attacks, but granulocytes from those patients still produced the greatest amount of PAF when stimulated with **calcium** ionophore A23187. (6) Although a major portion of **allergen**-induced PAF was secreted, less than 10% of ionophore-induced PAF was secreted. CONCLUSION: The findings that the circulating PAF increased markedly during acute asthmatic attacks and the enhanced in vivo and in vitro productions of PAF decreased to normal after successful immunotherapy strongly suggest that PAF may be involved in the pathogenesis of bronchial asthma.

L8 ANSWER 48 OF 59 MEDLINE
AN 93171500 MEDLINE
DN 93171500 PubMed ID: 8436775
TI Influence of oral **calcium** medication on nasal resistance in the nasal **allergen** provocation test.
AU Bachert C; Drechsler S; Hauser U; Imhoff W; Welzel D
CS Medical Department of Heinrich-Heine-University, Dusseldorf, Germany.
SO JOURNAL OF ALLERGY AND CLINICAL IMMUNOLOGY, (1993 Feb) 91 (2) 599-604. Journal code: 1275002. ISSN: 0091-6749.
CY United States
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199303
ED Entered STN: 19930402
Last Updated on STN: 19970203
Entered Medline: 19930322
TI Influence of oral **calcium** medication on nasal resistance in the nasal **allergen** provocation test.
AB Although **calcium** has been used for several decades to treat **allergic** diseases of the skin and respiratory tract, **controlled** studies demonstrating the action of oral preparations in **allergic** rhinitis are lacking. This placebo-**controlled**, double-blind, crossover study shows that 1000 mg **calcium** administered orally significantly inhibits the **allergen**-induced swelling of the nasal mucosa in the **allergen** provocation test. Sneezing and secretion, which are **allergic** symptoms, were not **reduced**. This study is the first to confirm the positive effect of oral **calcium** on a symptom of **allergic** rhinitis.
CT Check Tags: Female; Human; Male
Administration, Oral
Adolescence
Adult
*Airway Resistance: DE, drug effects
***Allergens: IM, immunology**
*Calcium: AD, administration & dosage
Calcium: BL, blood
Double-Blind Method
*Nasal Mucosa: DE, drug effects
*Nasal Provocation Tests
Pulmonary Ventilation: DE, drug effects
CN 0 (**Allergens**)

L8 ANSWER 49 OF 59 MEDLINE
AN 91252774 MEDLINE
DN 91252774 PubMed ID: 1710368
TI The structural requirements of epitopes with IgE binding capacity

demonstrated by three major **allergens** from fish, egg and tree pollen.

AU Elsayed S; Apold J; Holen E; Vik H; Florvaag E; Dybendal T

CS Laboratory of Clinical Biochemistry, University Hospital, University of Bergen, Norway.

SO SCANDINAVIAN JOURNAL OF CLINICAL AND LABORATORY INVESTIGATION. SUPPLEMENT, (1991) 204 17-31. Ref: 65
Journal code: 2984789R. ISSN: 0085-591X.

CY Norway

DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)

LA English

FS Priority Journals

EM 199107

ED Entered STN: 19910728
Last Updated on STN: 19960129
Entered Medline: 19910709

TI The structural requirements of epitopes with IgE binding capacity demonstrated by three major **allergens** from fish, egg and tree pollen.

AB Three major **allergens** from cod fish, egg white and tree pollen, were characterized by studies on their **allergenic** and antigenic structures. The major **allergen** of cod fish, **Allergen** M "parvalbumins pI 4.75", is composed of 113 amino acid residues with a molecular weight of 12,328 daltons. It comprised three domains, AB, CD and EF, consisting of 3 helices interspaced by one loop. Each of the loops of the CD and EF domains each coordinates one Ca2+. The antigenicity and **allergenicity** of **Allergen** M was deduced from studying the modified protein and some particular synthetic peptides. Three sites were encompassing IgE binding epitopes namely peptides 33-44, 65-74 and 88-96. A novel peptide (49-64), of the CD-domain, was demonstrated to be **allergenically**/antigenically active and cross reactive with birch **pollen allergen**, which incidentally was used as a negative **control**. This site encompassed two repetitive sequences (D-E-D-K) and (D-E-L-K), suggested to be mutually critical for the specificity of antibody binding. This hypothesis was reconfirmed by SPPS of several analogous peptides of region 39-64. Furthermore, peptide 88-103 of the EF-domain was similarly synthesized; it functioned as a monovalent hapten, blocking and not eliciting **allergic** reaction. Moreover, peptide 13-32 of domain AB, the non-**calcium** binding domain, was thoroughly tested. The results of PK inhibition showed clear activity and the peptide was found to function at the level of a divalent determinant. Ovalbumin (OA) is the most dominant of five major **allergens** of egg white and universally used as model protein. OA **allergenic** epitopes were shown to be mainly determined by the primary structure and depend on certain peptide chain length. The N-terminal decapeptide (OA 1-10) was shown to react with reaginic IgE. Direct skin test on egg **allergic** patients, showed no activity and the site was therefore concluded to encompasses one single Ig binding haptenic epitope. Peptide OA 323-339, was demonstrated to be valuable in studies of T-cell recognition of protein antigens. Three analogous peptides of this region were prepared and clearly shown to be immunogenic in rabbits and to bind specific IgE from patients **allergic** to egg. OA 323-339 was concluded to encompass an **allergenic** and antigenic epitope which was recognized by human and rabbit B-lymphocytes. Eight peptides in the region 11-122 were similarly synthesized. A test battery was performed to study this region using rabbit polyclonal antibodies and human specific IgE. Some of these sites were involved in binding of particular Ig paratopes. Five immunogenic peptides from the major **allergens** of tree pollen extracts (segment 23-38), were synthesized. The selection of those peptides was setteled using two algorithms for providing the optimal hydrophobicity.(ABSTRACT TRUNCATED AT 400 WORDS)

CT Check Tags: Animal; Support, Non-U.S. Gov't
***Allergens: CH, chemistry**
Allergens: IM, immunology
Amino Acid Sequence
*Egg White
*Epitopes: CH, chemistry
Epitopes: IM, immunology
*Fishes: IM, immunology
*Immunoglobulin E: IM, immunology
Molecular Sequence Data
Ovalbumin: CH, chemistry
Ovalbumin: IM, immunology
*Pollen: IM, immunology
CN 0 (**Allergens**); 0 (Epitopes)

L8 ANSWER 50 OF 59 MEDLINE
AN 91228130 MEDLINE
DN 91228130 PubMed ID: 1851340
TI Effect of a 5-lipoxygenase inhibitor on leukotriene generation and airway responses after **allergen** challenge in asthmatic patients.
AU Hui K P; Taylor I K; Taylor G W; Rubin P; Kesterson J; Barnes N C; Barnes P J
CS Department of Thoracic Medicine, National Heart and Lung Institute, London.
SO THORAX, (1991 Mar) 46 (3) 184-9.
Journal code: 0417353. ISSN: 0040-6376.
CY ENGLAND: United Kingdom
DT (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Priority Journals
EM 199106
ED Entered STN: 19910630
Last Updated on STN: 19970203
Entered Medline: 19910613
TI Effect of a 5-lipoxygenase inhibitor on leukotriene generation and airway responses after **allergen** challenge in asthmatic patients.
AB The effect of a single oral dose (800 mg) of zileuton (A-64077), a specific 5-lipoxygenase inhibitor, on the early and late airway responses to inhaled **allergen** was studied in a randomised, double blind, placebo **controlled**, and crossover trial in nine subjects with atopic asthma. Leukotriene generation was also assessed in vivo by measuring urinary leukotriene (LT) E4 excretion, and ex vivo by measuring **calcium** ionophore stimulated whole blood LTB4 production. Zileuton almost completely inhibited ex vivo LTB4 production but reduced urinary excretion of LTE4 by only about half. There was a trend for the early asthmatic response to be less on the day of zileuton treatment, but this did not reach statistical significance ($p = 0.08$). The zileuton induced **reduction** in maximum fall in FEV1 in the early asthmatic response was, however, significantly related to the **reduction** in urinary LTE4 excretion ($r = 0.8$), but not to the **reduction** in LTB4 generation ex vivo. There was no significant change in the **allergen** induced late asthmatic response, or in the increase in airway responsiveness to methacholine following antigen. The results provide some support for the hypothesis that the cysteinyl leukotrienes have a role in the **allergen** induced early asthmatic response. More complete in vivo inhibition of 5-lipoxygenase may be needed to produce a significant **reduction** in airway response to **allergen** challenge.
CT Check Tags: Human; Male
Adult
Allergens: IM, immunology

*Arachidonate 5-Lipoxygenase: AI, antagonists & inhibitors
Asthma: BL, blood
*Asthma: IM, immunology
Double-Blind Method
*Forced Expiratory Volume: DE, drug effects
*Hydroxyurea: AA, analogs & derivatives
Hydroxyurea: PD, pharmacology
Leukotriene B4: BL, blood
Leukotriene E4
*SRS-A: AA, analogs & derivatives
SRS-A: UR, urine
Time Factors

CN 0 (**Allergens**); 0 (SRS-A); EC 1.13.11.34 (Arachidonate 5-Lipoxygenase)

L8 ANSWER 51 OF 59 MEDLINE
AN 91182186 MEDLINE
DN 91182186 PubMed ID: 2080949
TI [**Reduction** of reactivity to **allergic** rhinitis with intravenous administration of **calcium**. Clinical-experimental study on the effect of changes of local airway resistance after nasal **allergen** provocation].
Verminderung der Reaktivitat bei Rhinitis **allergica** durch intravenose Applikation von Kalzium. Klinisch-experimentelle Studie uber die Beeinflussung des Atemwegswiderstandes nach nasaler **Allergen** -Provokation.
AU Bachert C; Drechsler S; Keilmann A; Seifert E; Schmidt R; Welzel D
CS Klinikum Mannheim Universitat Heidelberg.
SO ARZNEIMITTEL-FORSCHUNG, (1990 Sep) 40 (9) 984-7.
Journal code: 0372660. ISSN: 0004-4172.
CY GERMANY: Germany, Federal Republic of
DT (CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA German
FS Priority Journals
EM 199104
ED Entered STN: 19910519
Last Updated on STN: 19980206
Entered Medline: 19910429
TI [**Reduction** of reactivity to **allergic** rhinitis with intravenous administration of **calcium**. Clinical-experimental study on the effect of changes of local airway resistance after nasal **allergen** provocation].
Verminderung der Reaktivitat bei Rhinitis **allergica** durch intravenose Applikation von Kalzium. Klinisch-experimentelle Studie uber die Beeinflussung des Atemwegswiderstandes nach nasaler **Allergen** -Provokation.
AB The antiallergic activity of calcium was investigated in 25 patients with **allergic** rhinitis by nasal provocation with increasing doses of phleum pratense during a symptom free interval. Prior to that provocation, the patients received 9 mmol **calcium** (**Calcium**-Sandoz) i.v. or placebo respectively (double blind cross-over design). The concentration of serum **calcium** increased after **calcium** injection by 0.45 +/- 0.055 mmol/l. **Calcium** exerted a significant protective effect as compared to placebo: higher **allergen** doses, (p = 0.021) i.e. 20433 biological units/ml vs. 7494 biological units/ml, were required in order to induce a defined **allergic** reaction (50% decrease of nasal air flow). The data thus furnish evidence that intravenous **calcium** **reduces** the **allergic** response in type I **allergy**.

L8 ANSWER 52 OF 59 MEDLINE
AN 90166121 MEDLINE
DN 90166121 PubMed ID: 2624666
TI The effect of immunotherapy on the in vitro productions of histamine, prostaglandin E2 and leukotriene C4 in asthmatic children.
AU Wang J Y; Hsieh K H
CS Department of Pediatrics, College of Medicine, National Chengkung University Hospital, Tainan, Republic of China.
SO ASIAN PACIFIC JOURNAL OF ALLERGY AND IMMUNOLOGY, (1989 Dec) 7 (2) 119-24. Journal code: 8402034. ISSN: 0125-877X.
CY Thailand
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199004
ED Entered STN: 19900601
Last Updated on STN: 19900601
Entered Medline: 19900409
AB In order to elucidate the working mechanisms of immunotherapy (IT), the in vitro productions of histamine, prostaglandin E2 (PGE2) and leukotriene C4 (LTC4) were studied in 18 newly diagnosed and 20 hyposensitized (greater than 2 yr) asthmatic children. All were sensitive to house dust and dust **mites**. (D. pteronyssinus). Ten age-matched normal children were included as **control**. Polymorphonuclear (PMNs) and mononuclear (MNCs) leukocytes were separated by density gradient centrifugation and dextran sedimentation. PMNs (2 x 10(7) cells/ml) and MNCs (2 x 10(7) cells/ml) were stimulated with **mite allergen** (10 micrograms/ml) and **calcium** ionophore A23187 (1 microgram/ml) for 15 minutes. The plasma and culture supernatant (sup) histamine levels and sup PGE2 and LTC4 were measured by RIA. The results showed; 1) When compared to new patients, the treated patients had much lower plasma and sup histamine (p less than 0.001), no matter whether PMNs and MNCs were stimulated with **allergen** or A23187 and the normals had the lowest histamine level among 3 groups; 2) LTC4 in A23187-stimulated sup was lower in treated patients (p less than 0.05); 3) The PGE2 in **allergen**-stimulated sup was markedly increased in treated patients as compared to new patients (p less than 0.01) and the PGE2 in sup of normals was also much higher than that of new patients. Thus, immunotherapy is able to reverse the abnormal secretory pattern of inflammatory mediators of **allergic** patients, and this change may account, partly, for its clinical effectiveness.

L8 ANSWER 53 OF 59 MEDLINE
AN 89390448 MEDLINE
DN 89390448 PubMed ID: 2571293
TI Bronchial effects of alpha 2-adrenoceptor agonists and of other antihypertensive agents in asthma.
AU Dinh Xuan A T; Lockhart A
CS Physiology Laboratory, Cochin Port-Royal School of Medicine, Cochin Hospital, Paris, France.
SO AMERICAN JOURNAL OF MEDICINE, (1989 Sep 18) 87 (3C) 34S-37S. Ref: 45 Journal code: 0267200. ISSN: 0002-9343.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 198910
ED Entered STN: 19900309
Last Updated on STN: 19950206
Entered Medline: 19891023
AB The respective prevalence of hypertension and asthma is sufficient for

their combined existence to be far from rare. The effects of certain antihypertensive drugs, e.g., alpha 2-adrenoceptor agonists, on the bronchi may be either harmful or beneficial. When inhaled, alpha 2-agonists **reduce** the immediate bronchial response to **allergens**, whereas when ingested they aggravate the bronchial response to histamine and all the more so when their effect on the central nervous system is greater. Therefore, there has been much interest in agents such as the new oxazoline derivative, rilmenidine, which has less central effects than clonidine, an imidazoline compound of reference. **Calcium** antagonists inhibit smooth muscle contraction and release of mast cell inflammatory mediators. In asthmatic subjects, their short-term administration leads to a modest improvement in spontaneous bronchial obstruction, has only a partial protective action against various nonspecific or **allergenic** stimuli, and slightly reinforces the beneficial effect of beta 2-agonists. Beta-adrenoceptor antagonists aggravate bronchial obstruction and nonspecific bronchial hyperreactivity in asthmatic subjects. These harmful effects are dose-dependent, have even been reported after the administration of eyedrops, and are common to all beta-blockers. Angiotensin-converting enzyme inhibitors increase bronchial hyperreactivity in patients who develop cough during treatment and may, in certain cases, worsen or even induce asthma, probably by opposing inactivation by hydrolysis of tachykinins and of bradykinins.

L8 ANSWER 54 OF 59 MEDLINE
AN 87266962 MEDLINE
DN 87266962 PubMed ID: 2440260
TI Membrane sialic acid influences basophil histamine release by interfering with calcium dependence.
AU Jensen C; Norn S; Skov P S; Dahl B T; Thastrup O; Leon A; Svendsen U G
SO AGENTS AND ACTIONS, (1987 Apr) 20 (3-4) 161-4.
Journal code: 0213341. ISSN: 0065-4299.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198707
ED Entered STN: 19900305
Last Updated on STN: 19900305
Entered Medline: 19870729
AB The influence of the cell membrane content of sialic acid on basophil histamine release was examined in vitro in **allergic** patients and normal **controls**. Enzymatical removal of sialic acid enhanced histamine release induced by **allergen** and anti-IgE, whereas an increase in membrane sialic acid content by insertion of sialic acid containing gangliosides into the membrane inhibited the mediator release. The **reduction** in membrane sialic acid content abolished the inhibitory capacity of the **calcium** channel antagonist nimodipine, whereas the inhibition produced by verapamil and lanthanum was not affected. This difference, together with the previous finding that alterations in membrane sialic acid content is reflected in the cell sensitivity to extracellular calcium, suggest an interaction between membrane sialic acid and the calcium channels involved in basophil histamine release.

L8 ANSWER 55 OF 59 MEDLINE
AN 87107946 MEDLINE
DN 87107946 PubMed ID: 2433224
TI Adverse effects of acetylcysteine on human and guinea pig bronchial asthma in vivo and on human fibroblasts and leukocytes in vitro.
AU Dorsch W; Auch E; Powerlowicz P
SO INTERNATIONAL ARCHIVES OF ALLERGY AND APPLIED IMMUNOLOGY, (1987) 82 (1) 33-9.

Journal code: 0404561. ISSN: 0020-5915.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198702
ED Entered STN: 19900302
Last Updated on STN: 19970203
Entered Medline: 19870226
AB The term '**allergen** tachyphylaxis' denotes decreasing bronchial reactivity to **allergen** after repeated **allergen** inhalation challenges. In guinea pig bronchial asthma this self-protecting mechanism depends on endogenous prostaglandin E biosynthesis and can be inhibited by certain thiols. Therefore, we tested the effect of acetylcysteine (AC), a secretolytic thiol, on **allergen** tachyphylaxis in 25 guinea pigs. We observed inhibition of **allergen** tachyphylaxis and prolongation of each single asthmatic reaction. A possible clinical relevance of this observation was tested by the following experiments: Human lung fibroblasts (Wi-38) were stimulated with arachidonic acid and **calcium** ionophore and exposed to increasing amounts of AC. PgE biosynthesis was **reduced** from 2,408 pg/ml (**control**) to 84.2 pg/ml (0.6% AC) and 18.6 pg/ml (6% AC). Histamine release (HR) from human peripheral leukocytes was induced by anti-IgE. AC (0.016, 0.16, 1.6%) augmented both spontaneous HR (0-51.8%) and anti IgE induced HR (23.5-57.9%, p less than 0.001). Patients with isolated immediate bronchial reactions after **allergen** challenge inhaled 3 times a constant **allergen** dose. In few cases the reaction decreased from one test to another. This '**allergen** tachyphylaxis' was inhibited by AC. We conclude that AC should be used with caution in patients suffering from bronchial asthma.
CT Check Tags: Animal; Human; Male; Support, Non-U.S. Gov't
Acetylcysteine: AD, administration & dosage
*Acetylcysteine: AE, adverse effects
Administration, Inhalation
Allergens: PD, pharmacology
Asthma: CI, chemically induced
Asthma: IM, immunology
Fibroblasts: DE, drug effects
Guinea Pigs
Histamine Release
Leukocytes: DE, drug effects
Prostaglandins E: BI, biosynthesis
Tachyphylaxis
CN 0 (**Allergens**); 0 (Prostaglandins E)

L8 ANSWER 56 OF 59 MEDLINE
AN 87096926 MEDLINE
DN 87096926 PubMed ID: 3799404
TI [Bronchial hyperreactivity].
Hiperreactividad bronquial.
AU Rodriguez de la Vega A
SO ALLERGOLOGIA ET IMMUNOPATHOLOGIA, (1986 Sep-Oct) 14 (5) 363-7.
Journal code: 0370073. ISSN: 0301-0546.
CY Spain
DT Journal; Article; (JOURNAL ARTICLE)
LA Spanish
FS Priority Journals
EM 198701
ED Entered STN: 19900302
Last Updated on STN: 19900302
Entered Medline: 19870128
AB Bronchial hyperreactivity is a condition in which the airways show a much greater bronchoconstriction response to provocative stimuli than what is

normal. The stimuli may be specific (different **allergens**) or non-specific (exercise, infection, cold, air, ozone, kerosene or a variety of inhalant irritants). The normal **control** of the airways is regulated by: parasympathetic cholinergic nerves, sympathetic adrenergic nerves and non-adrenergic bronchodilator system. The activity in all these pathways regulates bronchomotor tone which is affected by many different reflexes. Such changes play a role in hyperreactivity. Exposure to **allergens** is another cause of inflammation and specific hyperreactivity which may increase the degree of non-specific bronchial reactivity. Inheritance has been implicated in bronchial hyperreactivity according to animal experiments and human twins studies. **Calcium** ions are involved in most cellular processes and their role in bronchial hyperreactivity is related to defects in calcium regulation and metabolism. Based on this speculation, calcium antagonist drugs have been used in the treatment of bronchial asthma, though no clinical improvement has been observed by most authors.

L8 ANSWER 57 OF 59 MEDLINE
AN 84102195 MEDLINE
DN 84102195 PubMed ID: 6660435
TI Intranasal verapamil in **allergen**-induced rhinitis.
AU Secher C; Brofeldt S; Mygind N
SO ALLERGY, (1983 Nov) 38 (8) 565-70.
Journal code: 7804028. ISSN: 0105-4538.
CY Denmark
DT (CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Priority Journals
EM 198402
ED Entered STN: 19900319
Last Updated on STN: 19980206
Entered Medline: 19840214
TI Intranasal verapamil in **allergen**-induced rhinitis.
AB Twenty-six **pollen-allergic** subjects participated in a double-blind, placebo-**controlled** trial of the protective effect of the **calcium** antagonist, verapamil, on **allergen** -provoked nasal symptoms. Intranasal verapamil, 1 mg. had a weak protective effect in that "tickling score" was 22% lower (P less than 0.01) and the number of sneezes 29% lower (nonsignificant) after verapamil as compared with placebo pretreatment. There were no differences with regard to nasal blockage or discharge. It is concluded that the verapamil spray used, cannot be recommended for clinical trials, but that further investigations of other formulations of calcium antagonist are justified in order to analyse the potential role of this type of drugs in the treatment of **allergic** rhinitis.
CT Check Tags: Female; Human; Male; Support, Non-U.S. Gov't
Administration, Intranasal
Adolescence
Adult
Airway Resistance: DE, drug effects
***Allergens: AD, administration & dosage**
Hay Fever: DI, diagnosis
*Hay Fever: DT, drug therapy
Hay Fever: ET, etiology
Nasal Provocation Tests
Placebos
Sneezing: DE, drug effects
Time Factors
Verapamil: AD, administration & dosage
Verapamil: AE, adverse effects

*Verapamil: TU, therapeutic use
CN 0 (**Allergens**); 0 (Placebos)

L8 ANSWER 58 OF 59 MEDLINE
AN 83202289 MEDLINE
DN 83202289 PubMed ID: 6303164
TI Effects of nifedipine on antigen-induced bronchoconstriction.
AU Henderson A F; Heaton R W; Dunlop L S; Costello J F
SO AMERICAN REVIEW OF RESPIRATORY DISEASE, (1983 May) 127 (5) 549-53.
Journal code: 0370523. ISSN: 0003-0805.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 198306
ED Entered STN: 19900318
Last Updated on STN: 19970203
Entered Medline: 19830617
AB We have investigated the effects of the **calcium** antagonist nifedipine on antigen-induced bronchoconstriction in vivo and in vitro. Eight grass-**pollen**-sensitive asthmatics were given either nifedipine (20 mg sublingually) or placebo 30 min before antigen challenge. The fall in forced expiratory volume in one second after pretreatment with placebo was 42.8 +/- 10.1%. After nifedipine this fall was significantly **reduced** to 26.5 +/- 11.7% (p less than 0.005). Two in vitro models of **allergic** asthma have been studied: actively sensitized guinea pig tracheal strips (GPT) and passively sensitized human bronchial muscle (HBM). Contraction of GPT by acetylcholine, histamine, and antigen challenge was unaffected by nifedipine 10(-4)M. Contraction of HBM by acetylcholine, histamine and grass **pollen** antigen challenge was significantly **reduced** by nifedipine 10(-4)M and 10(-6)M. The magnitude of the **reduction** in contraction to antigen challenge was comparable to the inhibition of acetylcholine and histamine responses. It would appear most likely that nifedipine exerts its effect mainly on bronchial muscle contractility rather than by stabilizing mast cells.

L8 ANSWER 59 OF 59 MEDLINE
AN 76239295 MEDLINE
DN 76239295 PubMed ID: 59779
TI Complement-mediated release of histamine from human basophils. II. Biochemical characterization of the reaction.
AU Grant J A; Settle L; Whorton E B; Dupree E
SO JOURNAL OF IMMUNOLOGY, (1976 Aug) 117 (2) 450-6.
Journal code: 2985117R. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 197610
ED Entered STN: 19900313
Last Updated on STN: 19970203
Entered Medline: 19761002
AB Release of histamine from human basophils was induced by activation of complement using zymosan. The histamine-releasing factor resembled C5a on the basis of m.w. (15,000) as well as previous studies showing **inactivation** by anti-C5. Complement-induced release of histamine was compared with **allergic** release of histamine which is mediated through appropriate **allergens** and reaginic IgE. Previously we demonstrated that the former reaction occurred more quickly. Both reactions were inhibited by drugs which increase intracellular concentrations of cAMP3 (theophylline, prostaglandin E1, and histamine) or which mimic the action of cAMP (its dibutyrly derivative). **Calcium**

was required for complement-mediated release of histamine and an increasing response was observed up to physiologic concentrations (2 mM). Magnesium (0 to 1 mM) did not affect the amount of histamine released. Also, glycolysis was probably required for optimal release by complement, since both 2-deoxyglucose and iodoacetamide were inhibitory. When basophils were partly enriched by depletion of neutrophils and eosinophils, the percentage of histamine released by complement was unchanged. Finally, it was shown that activated complement desensitized basophils from responding to a second challenge by the same stimulus. Cross-desensitization was not observed between complement and pollen **allergens.**

L9 ANSWER 39 OF 80 CAPLUS COPYRIGHT 2002 ACS
AN 1992:146172 CAPLUS
DN 116:146172
TI Thermoplastic composition containing mite repellents, for carpets
IN Onogaki, Kimiho; Onishi, Akiyoshi; Mori, Takashi
PA Mitsubishi Petrochemical Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 6 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 03153601	A2	19910701	JP 1989-291483	19891109
JP 2865742	B2	19990308		

AB A compn. consists of a thermoplastic resin 100, a light stabilizer 0.001-1.0, and I (R1 = OH, C1-9 hydrocarbyl; R2 = H, C1-30 hydrocarbyl, etc.; X = H, OH; n = 0-4) 0.1-10.0 parts by wt. This compn. is stable, wheathering resistant and useful for **controlling mites** in carpets, floor mats, blankets, etc. (no data). A mite repellent consisted of Ph salicylate 1, di-Me succinate condensation product with 1-(2-hydroxyethyl)-4-hydroxy-2,2,6,6-tetramethylpiperidine 0.1, M-329 (resin) 0.1, and Ca stearate 0.05 part by wt.
IT 1592-23-0, **Calcium** stearate 31566-31-1 36837-77-1
52829-07-9, LS-770 64022-61-3 71551-46-7, M329 71878-19-8
90751-07-8
RL: BIOL (Biological study)
(**mite**-repelling carpets contg.)

L9 ANSWER 37 OF 80 CAPLUS COPYRIGHT 2002 ACS
AN 1993:141858 CAPLUS
DN 118:141858
TI **Controlling** dust **mites** with powdered salts
IN Miller, Annette; Miller, Jeffrey D.
PA USA
SO PCT Int. Appl., 18 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9301722	A1	19930204	WO 1992-US6042	19920717
	W: AT, AU, BB, BG, BR, CA, CH, CS, DE, DK, ES, FI, GB, HU, JP, KP, KR, LK, LU, MG, MN, MW, NL, NO, PL, RO, RU, SD, SE, US				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, SN, TD, TG				
	US 5271947	A	19931221	US 1991-735063	19910724
	AU 9224088	A1	19930223	AU 1992-24088	19920717
PRAI	US 1991-735063		19910724		
	WO 1992-US6042		19920717		

TI **Controlling** dust **mites** with powdered salts
IT Carpets
(dust **mite control** in, with powd. salts)
IT Toys
(fabric-contg., dust **mite control** in, with powd. salts)
IT Acaricides
(powd. salts, for dust **mite control**)
IT Alkali metal chlorides
Alkaline earth chlorides
RL: BIOL (Biological study)
(powd., dust **mites control** by)
IT Household furnishings
(bedding, dust **mite control** in, with powd. salts)
IT Furniture
(upholstered, dust **mite control** in, with powd. salts)
IT 463-79-6D, Carbonic acid, alkali metal and alk. earth metal salts 497-19-8, Sodium carbonate, biological studies 7447-40-7, Potassium chloride, biological studies 7647-14-5, Sodium chloride, biological studies 7664-93-9D, Sulfuric acid, alkali metal and alk. earth metal salts 10028-22-5, Ferric sulfate 10043-52-4, **Calcium** chloride, biological studies 10294-66-3, Potassium thiosulfate 13686-28-7D, Thiosulfuric acid (H2S2O3), alkali metal and alk. earth metal salts
RL: BIOL (Biological study)
(powd., dust **mites control** by)

L9 ANSWER 8 OF 80 CAPLUS COPYRIGHT 2002 ACS
AN 2001:86201 CAPLUS
DN 134:127292
TI Dehumidifying **mite control** sheet containing moisture absorbents compartmentalized therein
IN Abe, Toshio; Kamimura, Satomi; Enomoto, Shoichi
PA Fumakilla Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 6 pp.
CODEN: JKXXAF
DT Patent
LA Japanese

Date no good for JP priority

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2001029730	A2	20010206	JP 1999-205320	19990719

TI Dehumidifying **mite control** sheet containing moisture absorbents compartmentalized therein
ST **mite control** moisture absorbent sheet compartmentalized package; silica gel moisture absorbent compartmentalized package **mite control**
IT Clays, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(activated; dehumidifying **mite control** sheet contg. moisture absorbents in compartmentalized package)
IT Acaricides
Colorimetric indicators
Drying agents
Hygroscopic substances
Insect repellents
(dehumidifying **mite control** sheet contg. moisture absorbents in compartmentalized package)
IT Diatomite
Silica gel, biological studies
Zeolites (synthetic), biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(dehumidifying **mite control** sheet contg. moisture absorbents in compartmentalized package)
IT Packaging materials
(films, gas-impermeable; dehumidifying **mite control** sheet contg. moisture absorbents in compartmentalized package)
IT Nonwoven fabrics
(packaging material; dehumidifying **mite control** sheet contg. moisture absorbents in compartmentalized package)
IT Absorbents
(water, polymers; dehumidifying **mite control** sheet contg. moisture absorbents in compartmentalized package)
IT Polymers, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(water-absorbing; dehumidifying **mite control** sheet contg. moisture absorbents in compartmentalized package)
IT 7440-44-0, Carbon, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(activated; dehumidifying **mite control** sheet contg. moisture absorbents in compartmentalized package)
IT 68-04-2, Sodium citrate 127-08-2, Potassium acetate 1305-78-8, **Calcium** oxide, biological studies 7558-79-4 7631-86-9, White carbon, biological studies 7786-30-3, Magnesium chloride, biological studies 10043-52-4, **Calcium** chloride, biological studies 63800-37-3, Sepiolite

RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(dehumidifying **mite control** sheet contg. moisture absorbents in compartmentalized package)

Date no good for JP Priority

L9 ANSWER 9 OF 80 CAPLUS COPYRIGHT 2002 ACS
AN 2001:85481 CAPLUS
DN 134:127291
TI Sheet-form house dust **mite control** agent containing hygroscopic substances
IN Abe, Toshio; Kamimura, Satomi; Enomoto, Shoichi; Shibata, Akinobu
PA Fumakilla Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 6 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2001029729	A2	20010206	JP 1999-205308	19990719

PI JP 2001029729 A2 20010206
TI Sheet-form house dust **mite control** agent containing hygroscopic substances
ST house dust **mite control** sheet hygroscopic substance; silica gel adhesion nonwoven fabric dust **mite control**
IT Clays, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(activated; sheet-form house dust **mite control** agents contg. hygroscopic substances to lower humidity for inhibiting growth)

IT Acaricides
Drying agents
Hygroscopic substances
Insect repellents
(sheet-form house dust **mite control** agents contg. hygroscopic substances to lower humidity for inhibiting growth)
IT Diatomite
Silica gel, biological studies
Zeolites (synthetic), biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(sheet-form house dust **mite control** agents contg. hygroscopic substances to lower humidity for inhibiting growth)
IT Absorbents
(water, polymers; sheet-form house dust **mite control** agents contg. hygroscopic substances to lower humidity for inhibiting growth)
IT Polymers, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(water-absorbing; sheet-form house dust **mite control** agents contg. hygroscopic substances to lower humidity for inhibiting growth)
IT 7440-44-0, Carbon, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(activated; sheet-form house dust **mite control** agents contg. hygroscopic substances to lower humidity for inhibiting growth)
IT 68-04-2, Sodium citrate 127-08-2, Potassium acetate 1305-78-8, **Calcium** oxide, biological studies 7558-79-4 7631-86-9, White carbon, biological studies 7786-30-3, Magnesium chloride, biological studies 10043-52-4, **Calcium** chloride, biological studies 63800-37-3, Sepiolite
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(sheet-form house dust **mite control** agents contg. hygroscopic substances to lower humidity for inhibiting growth)

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L20 ANSWER 1 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1999:678100 CAPLUS
DN 131:355169
TI Allergens in Paved Road Dust and Airborne Particles
AU Miguel, Ann G.; Cass, Glen R.; Glovsky, M. Michael; Weiss, Jay
CS Environmental Engineering Science Department, California Institute of Technology, Pasadena, CA, 91125, USA
SO Environmental Science and Technology (1999), 33(23), 4159-4168
CODEN: ESTHAG; ISSN: 0013-936X
PB American Chemical Society
DT Journal
LA English
RE.CNT 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
IT 7439-89-6, Iron, occurrence 7439-92-1, Lead, occurrence 7439-96-5, Manganese, occurrence 7440-17-7, Rubidium, occurrence 7440-24-6, **Strontium**, occurrence 7440-32-6, Titanium, occurrence 7440-48-4, Cobalt, occurrence 7440-50-8, Copper, occurrence 7440-62-2, Vanadium, occurrence 7440-66-6, Zinc, occurrence 7440-70-2, Calcium, occurrence 7723-14-0, Phosphorus, occurrence 7726-95-6, Bromine, occurrence 7782-50-5, Chlorine, occurrence
RL: POL (Pollutant); OCCU (Occurrence)
(**allergens** in paved road dust and airborne particles)

L20 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1997:386167 CAPLUS
DN 127:94038
TI Contents of calcium, magnesium, barium, aluminum and **strontium** in serum of **allergic** asthma patients
AU Han, Ziying; Du, Xuewu; Jin, Xiaoyan; Zhu, Guizhi; Yang, Ying
CS First Affiliated Hosp., Baotou Med. Coll., Baotou, 014010, Peop. Rep. China
SO Guangdong Weiliang Yuansu Kexue (1996), 3(11), 53-55
CODEN: GWYKF3; ISSN: 1006-446X
PB Guangdong Weiliang Yuansu Kexue Bianjibu
DT Journal
LA Chinese
TI Contents of calcium, magnesium, barium, aluminum and **strontium** in serum of **allergic** asthma patients
ST **allergic** asthma calcium magnesium barium aluminum; **strontium allergic** asthma
IT Asthma
(**allergic**; contents of calcium, magnesium, barium, aluminum and **strontium** in serum of **allergic** asthma patients)
IT 7429-90-5, Aluminum, biological studies 7439-95-4, Magnesium, biological studies 7440-24-6, **Strontium**, biological studies 7440-39-3, Barium, biological studies 7440-70-2, Calcium, biological studies
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(contents of calcium, magnesium, barium, aluminum and **strontium** in serum of **allergic** asthma patients)

L20 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1997:366286 CAPLUS
DN 126:334212
TI Cosmetic and pharmaceutical compositions containing salts of lanthanide, tin, zinc, manganese, yttrium, cobalt, strontium as substance P antagonists
IN Breton, Lionel; De Lacharriere, Olivier
PA Oreal S. A., Fr.
SO Eur. Pat. Appl., 10 pp.

CODEN: EPXXDW
DT Patent
LA French
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 770392	A2	19970502	EP 1996-402182	19961014
	EP 770392	A3	19970507		
	R: AT, BE, CH, DE, ES, FR, GB, IE, IT, LI, NL, SE				
	FR 2740335	A1	19970430	FR 1995-12658	19951026
	FR 2740335	B1	19971219		
	NO 9604517	A	19970428	NO 1996-4517	19961024
	JP 09165341	A2	19970624	JP 1996-284318	19961025
	JP 3112844	B2	20001127		
	CA 2188892	AA	19980425	CA 1996-2188892	19961025
	US 5900257	A	19990504	US 1996-738811	19961028
PRAI	FR 1995-12658	A	19951026		

IT **Allergy**
Drug delivery systems
Erythema
Eye, disease
Inflammation
Iridaceae
Pancreas, disease
Skin, disease
(cosmetic and pharmaceutical compns. contg. salts of lanthanide, tin, zinc, manganese, yttrium, cobalt, **strontium** as substance P antagonists)

L20 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1991:654048 CAPLUS
DN 115:254048
TI Effects of metal elements on .beta.-hexosaminidase release from rat basophilic leukemia cells (RBL-2H3)
AU Tanaka, Yukio; Takagaki, Yutaka; Nishimune, Takahiro
CS Osaka Prefect. Inst. Public Health, Osaka, 537, Japan
SO Chem. Pharm. Bull. (1991), 39(8), 2072-6
CODEN: CPBTAL; ISSN: 0009-2363
DT Journal
LA English
IT 1303-28-2, Arsenic oxide (As2O5) 1310-53-8, Germanium oxide (GeO2), biological studies 1327-53-3, Arsenic oxide (As2O3) 7446-18-6 7447-39-4, Copper chloride (CuCl2), biological studies 7447-41-8, Lithium chloride (LiCl), biological studies 7487-94-7, Mercury chloride (HgCl2), biological studies 7631-95-0 7646-79-9, Cobalt chloride (CoCl2), biological studies 7646-85-7, Zinc chloride (ZnCl2), biological studies 7647-17-8, Cesium chloride, biological studies 7718-54-9, Nickel chloride (NiCl2), biological studies 7720-78-7 7761-88-8, Nitric acid silver(1+) salt, biological studies 7773-01-5, Manganese chloride (MnCl2) 7789-00-6 7790-86-5, Cerium chloride (CeCl3) 7791-11-9, Rubidium chloride (RbCl), biological studies 10024-93-8, Neodymium chloride (NdCl3) 10025-73-7, Chromium chloride (CrCl3) 10099-58-8, Lanthanum chloride (LaCl3) 10099-74-8 10102-18-8 10108-64-2, Cadmium chloride (CdCl2) 10138-52-0, Gadolinium chloride (GdCl3) 10138-62-2, Holmium chloride (HoCl3) 10143-38-1 10168-80-6 10361-37-2, Barium chloride, biological studies 10361-82-7, Samarium chloride (SmCl3) **10476-85-4, Strontium chloride** 13410-01-0 13473-90-0 13510-49-1 13718-26-8 16903-35-8 16941-12-1
RL: BIOL (Biological study)
(.beta.-hexosaminidase release by basophil response to, as index of mediator release in immediate **allergy**)

L20 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1981:43371 CAPLUS
DN 94:43371
TI Improved methods for measuring radioactive tracer accumulation and excretion by microarthropods, with application for a mite species. Tyrophagus longior (Acarina, Acaridae)
AU Abbott, D. T.; Crossley, D. A., Jr.
CS Dep. Entomol., Georgia Univ., Athens, GA, USA
SO Report (1980), DOE/EV/00641-38, 16 pp. Avail.: NTIS From: Energy Res. Abstr. 1980, 5(21), Abstr. No. 34171
DT Report
LA English
ST **mite** radioelement metab; **strontium** 85 metab Tyrophagus; chromium 51 metab Tyrophagus

L20 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1980:634040 CAPLUS
DN 93:234040
TI Improved methods for measuring radioactive tracer accumulation and excretion by microarthropods, with applications for the mite, Tyrophagus longior (Gervais) (Acarina:Acaridae)
AU Abbott, David T.; Crossley, D. A., Jr.
CS Dep. Entomol., Univ. Georgia, Athens, GA, 30602, USA
SO Ann. Entomol. Soc. Am. (1980), 73(4), 492-4
CODEN: AESAAI; ISSN: 0013-8746
DT Journal
LA English
ST radioelement metab microarthropod detn; arthropod chromium 51 metab; **mite strontium** 90 metab

L20 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1976:161233 CAPLUS
DN 84:161233
TI Studies on the leukemogenic and immunologic effects of radiostrontium (strontium-90) and x-rays in mice
AU Ito, Takaaki; Nagao, Kenji; Kawamura, Yuzuru; Yokoro, Kenjiro
CS Res. Inst. Nucl. Med. Biol., Hiroshima Univ., Hiroshima, Japan
SO ERDA Symp. Ser. (1976), 37(Radiat. Lymphatic. Syst.), 209-17
CODEN: ERDSDX
DT Journal
LA English
IT **Allergy**
(delayed hypersensitivity, **strontium**-90 and x-ray effect on)

L20 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1975:454059 CAPLUS
DN 83:54059
TI Skin tolerance of flame-resistant cloth made from poly(terephthaloyl)oxalic acid bis(amidrazone) (PTO)
AU Nesswetha, W.
CS Forschungsstelle Bekleidungsmed. Enka Glanzst., Kelsterbach, Ger.
SO Berufs-Dermatosen (1974), 22(1), 28-33
CODEN: BERUAG
DT Journal
LA German
AB Poly(terephthaloyl)oxalic acid bis(amidrazone) [31051-04-4] or its zinc chelate, zinc-copper chelate, calcium chelate, or **strontium** hydroxide chelate used to manuf. synthetic cloth had no toxic or **allergic** effects on normal or irritated human skin in vivo. Application of the sterilized cloth to wounds or scarified skin caused no irritation and did not impair the healing process.

L20 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2002 ACS

AN 1963:48080 CAPLUS
DN 58:48080
OREF 58:8214f-g
TI Correlation between radiation tolerance and nuclear surface area
AU Iversen, Simon
CS Royal Beatson Mem. Hosp., Glasgow, UK
SO Nature (1962), 195, 1216-17
DT Journal
LA Unavailable
IT **Allergy**
(**strontium** metabolism in)

L20 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1963:48079 CAPLUS
DN 58:48079
OREF 58:8214f
TI Effect of inflammation of subcutaneous cellular tissue and of sensitization on stronium-90 distribution in the organism
AU Kalistratova, V. S.
SO Med. Radiol. (1962), 7(No. 12), 56-7
DT Journal
LA Unavailable
IT **Allergy**
Inflammation
Inflammation
(**strontium** metabolism in)

L20 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2002 ACS
AN 1963:48078 CAPLUS
DN 58:48078
OREF 58:8214d-f
TI Blood coagulation disturbances in chronic (occupational) x-ray irradiation
AU Shevchenko, V. I.
SO Med. Radiol. (1962), 7(No. 12), 49-55
DT Journal
LA Unavailable
IT **Allergy**
(**strontium** metabolism in)

L20 ANSWER 12 OF 16 WPIDS (C) 2002 THOMSON DERWENT
AN 2002-224934 [28] WPIDS
CR 2000-126669 [11]; 2000-126670 [11]; 2000-126695 [11]; 2000-126704 [11]; 2000-126834 [11]; 2000-137017 [12]; 2000-137019 [12]; 2000-137030 [12]; 2000-137031 [12]; 2000-160622 [14]; 2000-160623 [14]; 2000-365416 [31]; 2001-015868 [02]; 2001-024697 [03]; 2001-040841 [05]; 2001-060891 [07]; 2001-112369 [12]; 2001-122672 [13]; 2002-433314 [46]
DNN N2002-172383 DNC C2002-068575
TI Fecal component sensor device e.g. for diagnosing gastrointestinal disease, has micro-chip with array of sensors for detecting health or nutritional markers in body waste or human skin.
DC B04 S03
IN FEDOSOV, Y I; KHOMIAKOV, O N; KRUCHININ, M L; ROE, D C
PA (PROC) PROCTER & GAMBLE CO
CYC 1
PI US 6342037 B1 20020129 (200228)* 17p
ADT US 6342037 B1 Provisional US 1998-90993P 19980629, CIP of US 1998-106225 19980629, CIP of US 1998-107561 19980629, Provisional US 1999-131049P 19990426, US 1999-342754 19990629
FDT US 6342037 B1 CIP of US 6149636, CIP of US 6186591
PRAI US 1999-342754 19990629; US 1998-90993P 19980629; US 1998-106225 19980629; US 1998-107561 19980629; US 1999-131049P 19990426
AB US 6342037 B UPAB: 20020722
NOVELTY - A micro-chip comprising an array of chemical, electrochemical,

biochemical or biological sensors, is provided for detecting health or nutritional markers such as heavy metal, radioactive substances, fat, enzymes in body waste or human skin. The sensor provides a visual indication signal to the care taker.

USE - For detecting health markers such as heavy metals e.g. lead, mercury, radioactive substances e.g. cesium, **strontium**, uranium etc., fats, enzymes e.g. trypsin, chymotrypsin lipase, lactose, amylase, lipase, etc., endogenous secretions, proteinaceous matter e.g. casts, mucous and microorganisms like pathogenic bacteria, parasites, viruses, fungi, worms, protozoa, etc., and nutritional markers such as calcium, vitamins e.g. thiamine, riboflavin, niacin, biotin, folic acid, pantothenic acids, ascorbic acid, vitamin E, etc., electrolytes e.g. sodium, potassium, chlorine, bicarbonate etc., fats, fatty acids, soaps e.g. calcium palmitate, amino acids, bile acids, salts, steroids and carbohydrates for detecting metabolic efficiency, nutrient deficiencies, nutrient absorption or malabsorption, food and drink intake, food **allergies**, food intolerance e.g. lactose intolerance, colonic bacteria ecology e.g. bifidobacteria and lactobacillus for diagnosing various health issues such as infection, diarrhea, gastrointestinal disease, poisoning and skin irritation e.g. diaper dermatitis.

ADVANTAGE - Since the health and nutritional markers in body waste and human skin are detected, the caregivers and medical personnel can judge the condition of the patient efficiently.

DESCRIPTION OF DRAWING(S) - The figure shows an explanatory drawing of the sensor device.

Dwg.2/7

L20 ANSWER 13 OF 16 WPIDS (C) 2002 THOMSON DERWENT
AN 1998-245822 [22] WPIDS
DNN N1998-194533 DNC C1998-076908
TI Production of cloth capable of anti-bacterial treating - by screen printing anti-bacterial composition onto cloth.
DC D22 E12 F06 P74
PA (INUI-I) INUI K; (KANA-I) KANAYA K
CYC 1
PI JP 10077578 A 19980324 (199822)* 5p
JP 3291713 B2 20020610 (200241) 5p
ADT JP 10077578 A JP 1996-248566 19960830; JP 3291713 B2 JP 1996-248566 19960830
FDT JP 3291713 B2 Previous Publ. JP 10077578
PRAI JP 1996-248566 19960830
AB JP 10077578 A UPAB: 19980604
The cloth (CL) used for treating surface of a cloth (CS) to impart anti-bacterial property onto (CS) is prepared by printing an anti-bacterial compsn. (AC) onto (CS) by means of screen printing method (SP). (AC) is prepared by mixing **strontium** titanate or barium titanate, titanium oxide, aluminium oxide, metal salt of an amino-acid of L-glutaminic acid bound with acyl-gp. (of formula: RCO-; R=C11H23 to C17H35) from a natural fatty acid onto amino-gp. of the amino-acid, ceramics of a photo- semiconductor, an inorganic bactericide, an aq. binder, etc.

ADVANTAGE - The conventional method of impregnating organic bactericide,e.g. medical soap cannot be used for human skin with strong **allergy**, but present method can solve the problem.

Dwg.0/0

L20 ANSWER 14 OF 16 MEDLINE
AN 96340356 MEDLINE
DN 96340356 PubMed ID: 8747801
TI The Na+/K(+)-pump in rat peritoneal mast cells: some aspects of regulation of activity and cellular function.
AU Knudsen T
CS Department of Pharmacology, University of Odense.

SO DANISH MEDICAL BULLETIN, (1995 Nov) 42 (5) 441-54. Ref: 261
Journal code: 0066040. ISSN: 0907-8916.
CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 199609
ED Entered STN: 19961008
Last Updated on STN: 19961008
Entered Medline: 19960926
AB The mast cell contains potent mediators of inflammation which are released after IgE-directed and non-IgE-directed stimulation of the cell. This highly specialized cell is therefore ascribed a role in the pathogenesis of disease states in which the inflammatory response plays a role for the development of the clinical symptoms. Thus, besides being of interest in basic research, studies of the cellular processes leading to release of inflammatory mediators from the mast cell also have important clinical implications. The aim of the present work has been to document the existence of the Na+/K(+)-pump in rat peritoneal mast cells, to investigate the regulation of the pump activity and to explore whether modulation of the pump activity interferes with the cellular stimulus/secretion coupling mechanism. The Na+/K(+)-pump activity following stimulation of the mast cell was also investigated. The pump activity was assessed as the ouabain-sensitive cellular potassium uptake with 86Rb+ as a tracer for potassium. The histamine release from the mast cell following IgE-directed and non-IgE directed stimulation of the cell was used as a parameter for cellular degranulation. Histamine was measured by spectrofluorometry. The finding of an ouabain-sensitive uptake mechanism in the mast cell documents the presence of a functional Na+/K(+)-pump in this cell. The pump activity is inhibited by lanthanides and by the divalent cations calcium, magnesium, barium and **strontium**. The pump has a large reserve capacity which probably is caused by a low intracellular concentration of sodium. This enables the pump to respond to changes in the intracellular sodium concentration. The inhibitory effect of di- and trivalent ions on the pump activity is probably a result of the inhibitory effect of these ions on the cellular sodium uptake. The digitalis glycosides, ouabain and digoxin, but not the more lipophilic drug digitoxigenin, increase both IgE-directed and non-IgE-directed histamine release from the mast cell in a calcium-free medium, while there is no effect of digitalis glycosides in a medium containing physiologically relevant concentrations of calcium. The effect of digitalis glycosides on the histamine release is dependent on the drug concentrations used and the time of preincubation. An increase in the intracellular concentration of sodium secondary to inhibition of the Na+/K(+)-pump is the effector mechanism likely to explain the effect of digitalis glycosides on the mast cell histamine release. Increases in intracellular sodium might affect the intracellular concentration of calcium via changes in Na+/Ca(2+)-exchange. IgE-directed and non-IgE-directed stimulation of the mast cell activates the Na+/K(+)-pump. In case of compound 48/80-induced histamine release, the pump is stimulated for at least 2 hr. It is proposed, that the poststimulatory activation of the Na+/K(+)-pump is due to increased cellular sodium uptake associated with the release process. This sodium uptake may occur via Na+/Ca(2+)-exchange, Na+/H(+)-exchange, Na+/K+/2Cl(-)-cotransport or a non-selective ion channel. Besides describing aspects of the function and regulation of the Na+/K(+)-pump in the rat peritoneal mast cells, this thesis points to the potential role of sodium transport mechanisms in mast cell physiology. Pharmacological manipulations of such transport mechanisms might in the future add to the treatment of **allergic** diseases.

L20 ANSWER 15 OF 16 MEDLINE
AN 86251852 MEDLINE
DN 86251852 PubMed ID: 2424961
TI Enhanced basophil histamine release to concanavalin A in allergic rhinitis.
AU Busse W W; Swenson C A; Sharpe G; Koschat M
NC AI 10404 (NIAID)
AI 15685 (NIAID)
SO JOURNAL OF ALLERGY AND CLINICAL IMMUNOLOGY, (1986 Jul) 78 (1 Pt 1) 90-7.
Journal code: 1275002. ISSN: 0091-6749.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 198608
ED Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19860813
AB It has been suggested that IgE-dependent basophil histamine release (HR) does not necessarily relate to the amount of cell-bound IgE and, therefore, basophil "releasability" must be considered an important factor in this secretory process. To compare an IgE-dependent basophil HR process in nonatopic subjects and patients with **allergic** rhinitis, concanavalin A (Con A) was used as a secretagogue to stimulate mediator secretion. In 1.0 mmol/L of calcium-containing buffer, basophil HR to Con A (3.0 mcg/ml) was 50.2 +/- 8.6% in patients with **allergic** rhinitis and only 10.1 +/- 3.9% in nonatopic subjects. To evaluate whether this enhanced HR might be related to increased membrane influx of calcium, the following strategy was followed. **Strontium** (3.0 and 10.0 mmol/L) enhances immunologic (IgE) release of basophil histamine. Although the mechanism for **strontium** enhancement is not established, **strontium** may pass through the membrane channel more easily than calcium to increase secretion. We reasoned that if the enhanced release of histamine to Con A was related to increased membrane permeability to calcium, stimulation of basophil histamine secretion in the presence of **strontium** would reduce this difference. In both nonatopic subjects and patients with **allergic** rhinitis, **strontium** (3.0 and 10.0 mmol/L) enhanced HR. Enhanced HR with **strontium** was greater with basophils from normal subjects than from subjects with **allergic** rhinitis. Whether our observations with **strontium** indicate that the enhanced histamine releasability to Con A in subjects with **allergic** rhinitis may, in part, be due to a greater influx of calcium after immunologic stimulation must await characterization of the **strontium** effect or direct measurements of calcium ion disposition.(ABSTRACT TRUNCATED AT 250 WORDS)

L20 ANSWER 16 OF 16 MEDLINE
AN 62178040 MEDLINE
DN 62178040
TI Evaluation of a chemical depilatory for preoperative preparation of five hundred fifteen surgical patients.
AU PRIGOT A; GARNES A L; NWAGBO U
SO Amer J Surg, (1962 Dec) 104 900-6.
DT Journal
LA English
FS OLDMEDLINE
EM 196312
ED Entered STN: 19990716
Last Updated on STN: 19990716
ST drug **allergy**; glycolates; hair removal; hydroxides; **strontium**

Background

Dustroy™ Anti-All rg n Spray

Back To *SAFE CARE®*

AVAILABLE TESTING INFORMATION

ABC Laboratories: Ready Biodegradability Test by the CO2 Evolution Method
Aquatic Toxicity

Allergen Control Services: Dust Mite Allergen Denaturation (Der p1)

IBT Reference Laboratory: Composition and Comparison Studies for Denaturing Allergens
Der p 1 – Dust Mite
Der f 1 – Dust Mite
Fel d 1 – Cat
Can f 1 – Dog
Bla g 1 – Cockroach

Shinto Fine Company, Ltd.: Trial Report on MITE-NIX – Allergen-Denaturing Effects
(MITE-NIX is a private label customer of ***GEMTEK® Products***)

Scientific Material Int'l, Inc.: Specification testing for Material Safety Data Sheet

EXECUTIVE SUMMARY

The following represents ***GEMTEK® Products***' overview of the anti-allergen market and its interpretation of the comparison testing performed by IBT Reference Laboratory.

According to the National Center for Health Statistics, 41 million Americans suffer from allergies or asthma. The numbers grow 10% each year. One out of every seven households is affected. Approximately 10 million visits to the doctor result in a principal diagnosis of asthma; over 8 million visits to doctors are attributed to allergic rhinitis (allergies).

The alarming rise in allergy and asthma symptoms has anti-allergen manufacturers pushing for a profit. In 1995, $4.2 billion was spent on allergy and asthma treatment, $2.2 million on allergy and asthma products. Experts agree that avoidance is the best way to minimize the allergen effect. The leaders in the anti-allergen industry manufacture products containing tannic acid (can stain and affects mucous membranes, if inhaled), benzyl benzoate (toxic) and boron (hazardous) -- until now.

In 1997, ***GEMTEK® Products*** entered the anti-allergen market with their product line as a natural progression to **"Safely Cleaning Planet Earth™"**. In general, the issue of allergies surfaced, by the overwhelming concern for consumers who seek alternative products because of their chemical sensitivities. Since the introduction of its first ***Dustroy™*** products, ***GEMTEK®*** continued to pursue the development of exciting, new technologies in the denaturing of protein allergens created by dust mites, cats and dogs. The introduction of the ***AllerSafe™*** Anti-Allergen product line brings consumers a new level of effectiveness without toxic or damaging chemistries.

To demonstrate the effectiveness of this powerful new chemistry, ***GEMTEK®*** commissioned a study to compare the leading anti-allergen products with our unique formulations.

In order to assist the consumer in their comparison of various anti-allergen products, ***GEMTEK® Products*** has prepared this report summarizing the results of protein allergen neutralization by Dr. Brock Williams of IBT Research Laboratory.

The comparative testing of the ***Dustroy™*** formulations against prevailing anti-allergen formulations such as tannic acid and benzyl benzoate are broken down into both inhibition phase and reactivity phase. In this manner, the reader is able to more fully understand the complexity of the allergenicity and the effectiveness of each product type.

Five key household protein allergens were selected including:

- Der p1 and Der f1 (dust mites)

- Can f1 (dog)
- Fel d1 (cat)
- Bla g1 (cockroach)

The comparison testing performed at IBT utilized both dust samples and liquid allergens in solution. No effort was made to apply these products to carpeting or upholstery for comparison because of the lack of uniformity of such materials and the distribution of allergen soils. The tests were conducted in a controlled laboratory setting for the most accurate test results possible. The products tested for comparison purposes included:

- ***Dustroy*™ AA** and ***Dustroy*™ aa** manufactured by ***GEMTEK® Products***: This new product chemistry was tested in two concentrations and specifically formulated to neutralize dust mite allergens as a stand-alone anti-allergen spray.
- ***Dustroy*™ EX** and ***Dustroy*™ EX2** manufactured by ***GEMTEK® Products***: This product was formulated as a stand-alone anti-allergen neutralizing spray and/or an additive to other product formulations such as carpet shampoo, laundry detergent, etc.
- Allersearch ADS manufactured by Alkaline Corporation: It is our understanding that this product is based on a traditional tannic-acid formulation.
- Allercare manufactured by SC Johnson. It is our understanding that this product is based on benzyl benzoate as a ready-to-use spray -- product recalled during test trials.

The following data generated by IBT serves to describe one facet of anti-allergen solutions. Specifically, the degree and effectiveness of these solutions to render protein allergens inactive. In summary, the test indicate the following results:

- ***Dustroy*™ EX** was shown to have denaturing effects on Fel d1 only in dust samples containing these allergens.
- ***Dustroy*™ EX2** was shown to have no denaturing effects on allergens in dust samples.
- AllerCare was shown to have no denaturing effects on Fel d1 or Der f1, but was only somewhat effective on Can f1 and Der p1 at high concentrations in dust samples containing these allergens.
- Allersearch ADS was shown to have denaturing effects on Fel d1, Can f1, Der p1 and Der f1 in dust samples containing these allergens.
- ***Dustroy*™ aa** was shown to have very effective denaturing effects on Der p1 and Der f1 in liquid samples containing these allergens.
- Allersearch ADS was not effective in denaturing these allergens in liquid samples.

The findings are important. Traditional agents such as tannic acid and benzyl benzoate are shown to be relatively ineffective in the neutralization of these proteins. Additionally, both tannic acid and benzyl benzoate are known to be toxic and in the case of tannic acid, highly unstable and proven to discolor in the presence of acids and sunlight.

The ***Dustroy*™ AA** and ***Dustroy*™ EX** formulations are the new generation of anti-allergen solutions that are highly adaptive and effective and consumer friendly for a wide range of household applications. Specific use instructions vary with each application.

Realizing that the consumer has not been well informed about the effective use of anti-allergen solutions by the chemical supplier in the past, ***GEMTEK® Products*** is seeking to empower the consumer by helping to explain the allergen mechanism present in various allergen dust, to reduce the presence of such allergens and the recurring nature of protein allergens in most homes.

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More on this topic

Relevant sites from HON's MedHunt:

Allergen

Allergy-Provoking Substance

Multimedia from HONselect:

Allergens

Allergen

Allergy-provoking substance

An **antigen** (substance that elicits an **antibody** response) is responsible for producing allergic reactions by inducing **IgE** formation. IgE antibodies, bound to **basophils** in circulation and **mast cells** in tissue, cause these cells to release chemicals when they come into contact with an allergen. These chemicals can cause injury to surrounding tissue - the visible signs of an allergy. An allergen can be almost anything which acts as an antigen to stimulate such an immune response.

Common allergens

- **Food**. The most common are **milk**, **fruit**, fish, eggs and nuts.
- **Pollen**, especially ragweed, which causes hayfever.
- **Mould** from plants and food, which are most likely to cause asthma.
- **House dust**, which contains **mites** as well as **dander** from housepets.
- Venom from **insects** (such as bees, wasps and mosquitoes) or scorpions.
- Plant Oils, especially **poison** ivy, oak or sumac.

Additionally, feathers, wool, dyes, cosmetics and perfumes may also act as allergens.

Allergen Background info 9/03

Absolute Environmental
Allergy Store at home

Open the windows and enjoy the spring air without the mold spores, pollens or dust. Try one of our MicroAirScreens.

The Learning Center

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What are allergies?
What is asthma ?
Animal dander Allergies ?
Dust mite allergies ?
House dust allergies ?
Mold allergies ?
Multiple Chemical Sensitivity (MCS) ?
Pollen allergies ?
More Information

Read all about allergies and what causes those allergic reactions in the information to the left. We tell you all about the enemy: molds, pet dander, dust mites, pollen, household dust and what to do to make your indoor environment better. If you would like a hard copy of the information, consider purchasing a copy of the book this text came from.

Click on the book cover to buy it.

Condition	Allergy Index					
Asthma	Bedding	HEPA air cleaners	Denaturing Products	Nebulizers	Peak flow meters	Steam inhaler
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MCS	HEPA vacuum cleaners	Household Cleaning Products	Water Purification Systems	Personal Care Products	A/C Filter	
Mold Allergy	Mold test kit	HEPA air cleaners	Mold Growth Inhibitor	HEPA vacuum cleaners	A/C Filter	Air duct cleaning
Pollen Allergy	HEPA air cleaners	Denaturing Products	HEPA vacuum cleaners	A/C Filter	Air duct cleaning	Dust mask

What are allergies ?

Understanding the immune system

Self and Non-self

The heart of the immune system is the ability to distinguish between self and non-self. Virtually every body cell carries molecules that identify it as self. The body's immune defenses do not normally attack tissues that carry a self-marker. When immune defenders encounter cells or organisms carrying molecules that say "foreign," the immune troops move quickly to eliminate the intruders. Any substance capable of triggering an immune response is called an antigen. Antigens can be a virus, a bacterium, a fungus, or a parasite. An antigen announces its foreignness by means of characteristic shapes called epitopes, which protrude from its surface.

Keeping Out Foreigners

The immune system stockpiles a tremendous arsenal of cells. In order to have room to match millions of possible foreign invaders, just a few of each type of antibody are stored. When an antigen appears, those matched cells multiply into a full-scale army. Antibodies belong to a family of large molecules known as immunoglobulins. Immunoglobulins are proteins, made up of chains of amino acids. Scientists have identified nine chemically distinct classes of human immunoglobulins (Ig). Each type plays a different role in the immune defense strategy. IgE, which under normally occurs only in trace amounts, is the villain in allergic reactions. Each IgE antibody is specific; one reacts against oak pollen, another against ragweed.

OOPS! False Alarm

The first time an allergy-prone person is exposed to an allergen, he or she makes large amounts of the corresponding IgE antibody. These IgE molecules attach to the surfaces of cells in the body. When an IgE antibody encounters its specific allergen, it signals the body to begin powerful chemical warfare. These chemicals include histamine, heparin, eosinophils, and neutrophils.

Your Nose Knows these Symptoms.....
Do You?

It's really warfare, but to you, it may appear as one or more of the following symptoms:

- Sneezing often accompanied by a runny or clogged nose
- Coughing
- Postnasal drip
- Itching eyes, nose, or throat
- Allergic shiners (dark circles under the eyes caused by increased blood flow near the sinuses)
- The "allergic salute" (in a child, persistent upward rubbing of the nose that causes a crease mark on the nose)
- Watering eyes
- Conjunctivitis (inflammation of the membrane that lines the eyelids, causing red-rimmed, swollen eyes, and crusting of the eyelids).

First The Diagnosis

People with allergy symptoms, such as the runny nose of allergic rhinitis, may at first suspect they have a cold--but the "cold" lingers on. It is important to see a doctor about any respiratory illness that lasts longer than a week or two. When it appears that the symptoms are caused by an allergy, you should see a physician who understands the diagnosis and treatment of allergies. If the patient's medical history indicates that the symptoms recur at the same time each year, the physician will work under the theory

that a seasonal allergen (like pollen) is involved. Properly trained specialists recognize the patterns of potential allergens common during local seasons and the association between these patterns and symptoms. The medical history suggests which allergens are the likely culprits. The doctor also will examine the mucous membranes, which often appear swollen and pale or bluish in persons with allergic conditions.

Skin Tests

Doctors use skin tests to determine whether a patient has IgE antibodies in the skin that react to a specific allergen. The doctor use diluted extracts from allergens such as dust mites, pollens, or molds commonly found in the local area. The extract of each kind of allergen is injected under the patient's skin or is applied to a tiny scratch or puncture made on the patient's arm or back. Skin tests are one way of measuring the level of IgE antibody in a patient. With a positive reaction, a small, raised, reddened area (called a wheal) with a surrounding flush (called a flare) will appear at the test site. The size of the wheal can give the physician an important diagnostic clue, but a positive reaction does not prove that particular pollen is the cause of a patient's symptoms. Although such a reaction indicates that IgE antibody to a specific allergen is present in the skin, respiratory symptoms do not necessarily result.

Blood Tests

Although skin testing is the most sensitive and least costly way to identify allergies in patients, some patients such as those with widespread skin conditions like eczema should not be tested using that method. There are other diagnostic tests that use a blood sample from the patient to detect levels of IgE antibody to a particular allergen. One such blood test is called the RAST (radioallergosorbent test), which can be performed when eczema is present or if a patient has taken medications that interferes with skin testing.

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What is asthma ?

Asthma is a reversible obstructive lung disease, caused by an increased reaction of the airways to various stimuli. It is a chronic condition with acute exacerbations. In this country, there are approximately 28 million asthmatics; nearly one third of them (8.6 million) are children under 18 years of age. Asthma can be a life-threatening disease if not properly managed. Asthma is characterized by excessive sensitivity of the lungs to various stimuli. Asthma breathing problems usually happen in "episodes" or "attacks". An asthma episode is a series of events that result in narrowed airways. These include: swelling of the lining , tighting of muscles, and increased secretion of mucus in the airway. The narrowed airway is responsible for the difficulty in breathing with the familiar "wheeze".Triggers range from viral infection to allergies, to irritating gases and particles in the air. Each person reacts differently to the factors that may trigger asthma, including some respiratory infections; colds; allergic reactions to pollen, mold, animal dander, feathers, dust food$_1$ and cockroaches; vigorous exercise; exposure to cold air or sudden temperature change; cigarette smoke; excitement, and stress.

Asthma therapy includes efforts to reduce the underlying inflammation and to relieve or prevent symptomatic airway narrowing. Such efforts should lead to reduction in airway hyperresponsiveness and help prevent irreversible airway obstruction

The two classes of medications used to treat asthma are bronchodilators and anti-inflammatory agents.

- Mti-inflammatory agents interrupt the development of bronchial inflammation and have a prophylactic or preventive action. They may also modulate or terminate ongoing inflammatory reaction in the airways. These agents include corticosteriods, cromolyn sodium or cromolyn-like compounds, and other anti-inflammatory compounds.
- Bronchodilators act principally to dilate the airways by relaxing bronchial smooth muscle. They include bet~adrenergic agonists, methylxanthines, and anticholinergics

Asthma is the leading serious chronic illness among children. Most children have mild to moderate problems and their illness can be controlled by treatment at home or in the doctor's office. For some children the illness becomes a formidable problem causing numerous visits to the hospital emergency room and multiple hospitalizations

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Animal Dander Allergy

Household pets are the most common source of allergic reactions to animals. Many people think that pet allergy is provoked by the fur of cats and dogs. But researchers have found that the major allergens are proteins secreted by oil glands in the animals' skin and shed in dander as well as proteins in the saliva, which sticks to the fur when the animal licks itself. People have always said that when it comes to allergies, cats are worse than dogs. We now know that it is because cats lick themselves more than dogs, thereby spreading the allergens. In addition, cats may be held more and spend more time in the house, close to humans. Urine is also a source of allergy-causing proteins. When the substance carrying the proteins dries, the proteins can then float into the air. Some rodents, such as guinea pigs and gerbils, have become increasingly popular as household pets. They, too, can cause allergic reactions in some people, as can mice and rats. Urine is the major source of allergens from these animals. Allergies to animals can take two years or more to develop and may not subside until six months or more after ending contact with the animal. Carpet and furniture are a reservoir for pet allergens, and the allergens can remain in them for four to six weeks. In addition, these allergens can stay in household air for months after the animal has been removed. Therefore, it is wise for people with an animal allergy to check with the landlord or previous owner to find out if furry pets had lived previously on the premises.

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House Dust & Dust Mite Allergy

An allergy to dust found in houses is perhaps the most common cause of perennial allergic rhinitis. House dust allergy usually produces symptoms similar to pollen allergy.

What is house dust?

Rather than a single substance, house dust is a varied mixture of potentially allergenic materials. The particles seen floating in a shaft of sunlight may contain fibers from different types of fabrics; cotton lint, feathers, and other stuffing materials; bacteria; mold and fungus spores (especially in damp areas); food particles; bits of plants and insects; and other allergens peculiar to an individual home. Dust also may contain microscopic mites. These mites also live in bedding, upholstered furniture, and carpets. Ordinarily, they would thrive in summer and die in winter. However, in a warm, humid house, they

continue to thrive even in the coldest months. These waste products, which are proteins, actually provoke the allergic reaction. House dust mite allergy is the major year-round allergy in the world, though ragweed is more prevalent in the United States. Waste products of cockroaches are also an important cause of allergy symptoms from household allergens, particularly in some urban areas of the United States.

What are Dust Mites?

Dust mites are tiny animals you cannot see. Every home has dust mites. They feed on skin flakes and are found in mattresses, pillows, carpets, upholstered furniture, bedcovers, clothes, stuffed toys, and fabric or other fabric-covered items. Body parts and feces of dust mites can trigger allergic reactions in sensitive individuals. The presence of dust mites in a home are in no way an indication of the sanitary conditions in the home.

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Mold Allergy

Along with pollens from trees, grasses, and weeds, molds are an important cause of seasonal allergic rhinitis. People allergic to molds may have symptoms from spring to late fall. The mold season often peaks from July to late summer. Unlike pollens, molds may persist after the first killing frost. Some can grow at subfreezing temperatures, but most become dormant. Snow cover lowers the outdoor mold count dramatically but does not kill molds. After the spring thaw, molds thrive on the vegetation that has been killed by the winter cold. In the warmest areas of the United States, however, molds thrive all year and can cause year-round (perennial) allergic problems. In addition, molds growing indoors can cause perennial allergic rhinitis even in the coldest climates.

What is mold?

There are thousands of types of molds and yeast, the two groups of plants in the fungus family. Yeasts are single cells that divide to form clusters. Molds consist of many cells that grow as branching threads called hyphae. Although both groups can probably cause allergic reactions, only a small number of molds are widely recognized offenders. The seeds or reproductive particles of fungi are called spores. They differ in size, shape, and color among species. Each spore that germinates can give rise to new mold growth, which in turn can produce millions of spores.

What is mold allergy?

When inhaled, microscopic fungal spores or, sometimes, fragments of fungi may cause allergic rhinitis. Because they are so small, mold spores may evade the protective mechanisms of the nose and upper respiratory tract to reach the lungs. In a small number of people, symptoms of mold allergy may be brought on or worsened by eating certain foods, such as cheeses, processed with fungi. Occasionally, mushrooms, dried fruits, and foods containing yeast, soy sauce, or vinegar will produce allergic symptoms. There is no known relationship, however, between a respiratory allergy to the mold *Penicillium* and an allergy to the drug penicillin, made from the mold.

Where do molds grow?

Molds can be found wherever there is moisture, oxygen, and a source of the few other chemicals they need. In the fall they grow on rotting logs and fallen leaves, especially in moist, shady areas. In gardens, they can be found in compost piles and on certain grasses and weeds. Some molds attach to grains such as wheat, oats, barley, and corn, making farms; grain bins, and silos likely places to find mold.

Hot spots of mold growth in the home include damp basements and closets, bathrooms (especially shower stalls), places where fresh food is stored, refrigerator drip trays, house plants, air conditioners, humidifiers, garbage pails, mattresses, upholstered furniture, and old foam rubber pillows. Bakeries, breweries, barns, dairies, and greenhouses are favorite places for molds to grow. Loggers, mill workers, carpenters, furniture repairers, and upholsterers often work in moldy environments.

Which molds are allergenic?

Like pollens, mold spores are airborne allergens that are abundant, easily carried by air currents, and allergenic in their chemical makeup. Found almost everywhere, mold spores in some areas are so numerous they often outnumber the pollens in the air. Fortunately, however, only a few dozen different types are significant allergens. In general, *Alternaria* and *Cladosporium (Hormodendrum)* are the molds most commonly found both indoors and outdoors throughout the United States. *Aspergillus, Penicillium, Helminthosporium, Epicoccum; Fusarium; Mucor; Rhizopus*, and *Aureobasidium (Pullularia)* are also common.

Are there other mold-related disorders?

Fungi or microorganisms related to them may cause other health problems similar to allergic diseases. Some kinds of *Aspergillus* may cause several different illnesses, including both infections and allergy. These fungi may lodge in the airways or a distant part of the lung and grow until they form a compact sphere known as a "fungus ball." In people with lung damage or serious underlying illnesses, *Aspergillus* may grasp the opportunity to invade the lungs or the whole body. In some individuals, exposure to these fungi also can lead to asthma or to a lung disease resembling severe inflammatory asthma called allergic bronchopulmonary aspergillosis. This latter condition, which occurs only in a minority of people with asthma, is characterized by wheezing, low-grade fever, and coughing up of brown-flecked masses or mucus plugs. Skin testing, blood tests, X-rays, and examination of the sputum for fungi can help establish the diagnosis. Corticosteroid drugs are usually effective in treating this reaction; immunotherapy (allergy shots) is not helpful.

Indoor Air Regulations and Mold

Standards or Threshold Limit Values (TLVs) for airborne concentrations of mold, or mold spores, have not been set. Currently, there are no EPA regulations or standards for airborne mold contaminants.

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Multiple Chemical Sensitivity

Synthetic chemicals are all around us. They're in the products we use, in the clothes we wear, in the food we eat, in the air we breathe at work. Because chemicals are everywhere in the environment, it's not

possible to escape exposure. No wonder, then, that many people have become sensitized to the chemicals around them. For some people the sensitization is not too serious a problem. They may have what appears to be a minor allergy to one or more chemicals. Chemical sensitivity is not a true allergic reaction because IgE is not actually present. Other people are much more seriously affected. They may feel tired all the time, and suffer from mental confusion, breathing problems, sore muscles, and a weakened immune system. Such people suffer from a condition referred to as Multiple Chemical Sensitivity (MCS).

What is Multiple Chemical Sensitivity?

MCS is a disorder triggered by exposures to chemicals in the environment. Individuals with MCS can have symptoms from chemical exposures at concentrations far below the levels tolerated by most people. Symptoms occur in more than one organ system in the body, such as the nervous system and the lungs. Exposure may be from the air, from food or water, or through skin contact. The symptoms may look like an allergy because they tend to come and go with exposures, though some people's reactions may be delayed. As MCS gets worse, reactions become more severe and increasingly chronic, often affecting more bodily functions. No single widely available medical test can explain symptoms. In the early stages of MCS, repeat exposure to the substance or substances that caused the initial health effects provokes a reaction. After a time, it takes less and less exposure to this or related chemicals to cause symptoms. As the body breaks down, an ever-increasing number of chemicals, including some unrelated to the initial exposure, are found to trigger a reaction. MCS affects the overall health and feeling of well being of those with the disorder. It typically impairs many bodily functions including the nervous system and digestion. Each individual affected by MCS has a unique set of health problems. A chemically sensitive person may also have other preexisting health conditions. Many affected people experience a number of symptoms, in relation to their chemical exposures. MCS may result from a single massive exposure to one or more toxic substance or repeated exposures to low doses. People with MCS may become partially or totally disabled for several years or for life.

Treatment

MCS is difficult for physicians to define and diagnose. There is no single set of symptoms which fit together as neither a syndrome, nor a single diagnostic test for MCS. Instead, physicians should take a complete patient history that includes environmental and occupational exposures, and act as detectives in diagnosing this problematic condition. After the onset of MCS, a person's health generally continues to deteriorate. It may only begin to improve once the chemical sensitivity condition is uncovered. While a number of treatments may help improve the baseline health status for some patients, at the present time, there is no single "cure" except avoidance.

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Pollen Allergy

Pollen

Each spring, summer, and fall, tiny particles are released from trees, weeds, and grasses. These particles, known as pollen, hitch rides on currents of air. Although their mission is to fertilize parts of other plants, many never reach their targets. Instead, they enter human noses and throats, triggering a type of seasonal allergic rhinitis called pollen allergy, which many people know as hay fever or rose fever (depending on

the season in which the symptoms occur). Of all the things that can cause an allergy, pollen is one of the most widespread. People with pollen allergies often develop sensitivities to other troublemakers that are present all year, such as dust mites. Year-round airborne allergens cause perennial allergic rhinitis, as distinguished from seasonal allergic rhinitis.

What is pollen?

Plants produce microscopic round or oval pollen grains to reproduce. In some species, the plant uses the pollen from its own flowers to fertilize itself. Other types must be cross-pollinated; that is, pollen must be transferred from the flower of one plant to that of another plant of the same species. Insects do this job for certain flowering plants, while other plants rely on wind transport. The types of pollen that most commonly cause allergic reactions are produced by the plain-looking plants (trees, grasses, and weeds) that do not have showy flowers. These plants manufacture small, light, dry pollen granules that are custom-made for wind transport.

Where is pollen most common?

Most allergenic pollen comes from plants that produce it in huge quantities. A single ragweed plant can generate a million grains of pollen a day. Samples of ragweed pollen have been collected 400 miles out at sea and 2 miles high in the air. The chemical makeup of pollen is the factor that determines whether it is likely to cause hay fever. For example, pine tree pollen is produced in large amounts by a common tree, which would make it a good candidate for causing allergy. The chemical composition of pine pollen, however, appears to make it less allergenic than other basic types. Because pine pollen is heavy, it tends to fall straight down and does not scatter. Therefore, it rarely reaches human noses. Among North American plants, weeds are the most prolific producers of allergenic pollen. Ragweed is the major culprit, but others of importance are sagebrush, redroot pigweed, lamb's quarters, Russian thistle (tumbleweed), and English plantain. Grasses and trees, too, are important sources of allergenic pollens. Although more than 1,000 species of grass grow in North America, only a few produce highly allergenic pollen. These include timothy grass, Kentucky bluegrass, Johnson grass, Bermuda grass, redtop grass, orchard grass, and sweet vernal grass. Trees that produce allergenic pollen include oak, ash, elm, hickory, pecan, box elder, and mountain cedar. It is common to hear people say that they are allergic to colorful or scented flowers. In fact, only florists, gardeners, and others who have prolonged, close contact with flowers are likely to become sensitized to pollen from these plants. Most people have little contact with the large, heavy, waxy pollen grains of many flowering plants because this type of pollen is not carried by wind but by insects such as butterflies and bees.

When do plants make pollen?

One of the most obvious features of pollen allergy is its seasonal nature--people experience it symptoms only when the pollen grains to which they are allergic are in the air. Each plant has a pollinating period that is more or less the same from year to year. Exactly when a plant starts to pollinate seems to depend on the relative length of night and day--and therefore on geographical location--rather than on the weather. (On the other hand, weather conditions during pollination can affect the amount of pollen produced and distributed in a specific year.) Thus, the farther North you go, the later the pollinating period and the later the allergy season. A pollen count, which is familiar to many people from local weather reports, is a measure of how much pollen is in the air. This count represents the concentration of all the pollen (or of one particular type, like ragweed) in the air in a certain area at a specific time. It is expressed in grains of pollen per square meter of air collected over 24 hours. Pollen counts tend to be highest early in the morning on warm, dry, breezy days and lowest during chilly, wet periods. Although a pollen count is an approximate and fluctuating measure, it is useful as a general guide for when it is

advisable to stay indoors and avoid contact with the pollen.

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that a seasonal allergen (like pollen) is involved. Properly trained specialists recognize the patterns of potential allergens common during local seasons and the association between these patterns and symptoms. The medical history suggests which allergens are the likely culprits. The doctor also will examine the mucous membranes, which often appear swollen and pale or bluish in persons with allergic conditions.

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Doctors use skin tests to determine whether a patient has IgE antibodies in the skin that react to a specific allergen. The doctor use diluted extracts from allergens such as dust mites, pollens, or molds commonly found in the local area. The extract of each kind of allergen is injected under the patient's skin or is applied to a tiny scratch or puncture made on the patient's arm or back. Skin tests are one way of measuring the level of IgE antibody in a patient. With a positive reaction, a small, raised, reddened area (called a wheal) with a surrounding flush (called a flare) will appear at the test site. The size of the wheal can give the physician an important diagnostic clue, but a positive reaction does not prove that particular pollen is the cause of a patient's symptoms. Although such a reaction indicates that IgE antibody to a specific allergen is present in the skin, respiratory symptoms do not necessarily result.

Blood Tests

Although skin testing is the most sensitive and least costly way to identify allergies in patients, some patients such as those with widespread skin conditions like eczema should not be tested using that method. There are other diagnostic tests that use a blood sample from the patient to detect levels of IgE antibody to a particular allergen. One such blood test is called the RAST (radioallergosorbent test), which can be performed when eczema is present or if a patient has taken medications that interferes with skin testing.

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What is asthma ?

Asthma is a reversible obstructive lung disease, caused by an increased reaction of the airways to various stimuli. It is a chronic condition with acute exacerbations. In this country, there are approximately 28 million asthmatics; nearly one third of them (8.6 million) are children under 18 years of age. Asthma can be a life-threatening disease if not properly managed. Asthma is characterized by excessive sensitivity of the lungs to various stimuli. Asthma breathing problems usually happen in "episodes" or "attacks". An asthma episode is a series of events that result in narrowed airways. These include: swelling of the lining , tighting of muscles, and increased secretion of mucus in the airway. The narrowed airway is responsible for the difficulty in breathing with the familiar "wheeze".Triggers range from viral infection to allergies, to irritating gases and particles in the air. Each person reacts differently to the factors that may trigger asthma, including some respiratory infections; colds; allergic reactions to pollen, mold, animal dander, feathers, dust food$_1$ and cockroaches; vigorous exercise; exposure to cold air or sudden temperature change; cigarette smoke; excitement, and stress.

Asthma therapy includes efforts to reduce the underlying inflammation and to relieve or prevent symptomatic airway narrowing. Such efforts should lead to reduction in airway hyperresponsiveness and help prevent irreversible airway obstruction

The two classes of medications used to treat asthma are bronchodilators and anti-inflammatory agents.

- Mti-inflammatory agents interrupt the development of bronchial inflammation and have a prophylactic or preventive action. They may also modulate or terminate ongoing inflammatory reaction in the airways. These agents include corticosteriods, cromolyn sodium or cromolyn-like compounds, and other anti-inflammatory compounds.
- Bronchodilators act principally to dilate the airways by relaxing bronchial smooth muscle. They include bet~adrenergic agonists, methylxanthines, and anticholinergics

Asthma is the leading serious chronic illness among children. Most children have mild to moderate problems and their illness can be controlled by treatment at home or in the doctor's office. For some children the illness becomes a formidable problem causing numerous visits to the hospital emergency room and multiple hospitalizations

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Animal Dander Allergy

Household pets are the most common source of allergic reactions to animals. Many people think that pet allergy is provoked by the fur of cats and dogs. But researchers have found that the major allergens are proteins secreted by oil glands in the animals' skin and shed in dander as well as proteins in the saliva, which sticks to the fur when the animal licks itself. People have always said that when it comes to allergies, cats are worse than dogs. We now know that it is because cats lick themselves more than dogs, thereby spreading the allergens. In addition, cats may be held more and spend more time in the house, close to humans. Urine is also a source of allergy-causing proteins. When the substance carrying the proteins dries, the proteins can then float into the air. Some rodents, such as guinea pigs and gerbils, have become increasingly popular as household pets. They, too, can cause allergic reactions in some people, as can mice and rats. Urine is the major source of allergens from these animals. Allergies to animals can take two years or more to develop and may not subside until six months or more after ending contact with the animal. Carpet and furniture are a reservoir for pet allergens, and the allergens can remain in them for four to six weeks. In addition, these allergens can stay in household air for months after the animal has been removed. Therefore, it is wise for people with an animal allergy to check with the landlord or previous owner to find out if furry pets had lived previously on the premises.

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House Dust & Dust Mite Allergy

An allergy to dust found in houses is perhaps the most common cause of perennial allergic rhinitis. House dust allergy usually produces symptoms similar to pollen allergy.

What is house dust?

Rather than a single substance, house dust is a varied mixture of potentially allergenic materials. The particles seen floating in a shaft of sunlight may contain fibers from different types of fabrics; cotton lint, feathers, and other stuffing materials; bacteria; mold and fungus spores (especially in damp areas); food particles; bits of plants and insects; and other allergens peculiar to an individual home. Dust also may contain microscopic mites. These mites also live in bedding, upholstered furniture, and carpets. Ordinarily, they would thrive in summer and die in winter. However, in a warm, humid house, they

continue to thrive even in the coldest months. These waste products, which are proteins, actually provoke the allergic reaction. House dust mite allergy is the major year-round allergy in the world, though ragweed is more prevalent in the United States. Waste products of cockroaches are also an important cause of allergy symptoms from household allergens, particularly in some urban areas of the United States.

What are Dust Mites?

Dust mites are tiny animals you cannot see. Every home has dust mites. They feed on skin flakes and are found in mattresses, pillows, carpets, upholstered furniture, bedcovers, clothes, stuffed toys, and fabric or other fabric-covered items. Body parts and feces of dust mites can trigger allergic reactions in sensitive individuals. The presence of dust mites in a home are in no way an indication of the sanitary conditions in the home.

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Mold Allergy

Along with pollens from trees, grasses, and weeds, molds are an important cause of seasonal allergic rhinitis. People allergic to molds may have symptoms from spring to late fall. The mold season often peaks from July to late summer. Unlike pollens, molds may persist after the first killing frost. Some can grow at subfreezing temperatures, but most become dormant. Snow cover lowers the outdoor mold count dramatically but does not kill molds. After the spring thaw, molds thrive on the vegetation that has been killed by the winter cold. In the warmest areas of the United States, however, molds thrive all year and can cause year-round (perennial) allergic problems. In addition, molds growing indoors can cause perennial allergic rhinitis even in the coldest climates.

What is mold?

There are thousands of types of molds and yeast, the two groups of plants in the fungus family. Yeasts are single cells that divide to form clusters. Molds consist of many cells that grow as branching threads called hyphae. Although both groups can probably cause allergic reactions, only a small number of molds are widely recognized offenders. The seeds or reproductive particles of fungi are called spores. They differ in size, shape, and color among species. Each spore that germinates can give rise to new mold growth, which in turn can produce millions of spores.

What is mold allergy?

When inhaled, microscopic fungal spores or, sometimes, fragments of fungi may cause allergic rhinitis. Because they are so small, mold spores may evade the protective mechanisms of the nose and upper respiratory tract to reach the lungs. In a small number of people, symptoms of mold allergy may be brought on or worsened by eating certain foods, such as cheeses, processed with fungi. Occasionally, mushrooms, dried fruits, and foods containing yeast, soy sauce, or vinegar will produce allergic symptoms. There is no known relationship, however, between a respiratory allergy to the mold *Penicillium* and an allergy to the drug penicillin, made from the mold.

Where do molds grow?

Molds can be found wherever there is moisture, oxygen, and a source of the few other chemicals they need. In the fall they grow on rotting logs and fallen leaves, especially in moist, shady areas. In gardens, they can be found in compost piles and on certain grasses and weeds. Some molds attach to grains such as wheat, oats, barley, and corn, making farms; grain bins, and silos likely places to find mold.

Hot spots of mold growth in the home include damp basements and closets, bathrooms (especially shower stalls), places where fresh food is stored, refrigerator drip trays, house plants, air conditioners, humidifiers, garbage pails, mattresses, upholstered furniture, and old foam rubber pillows. Bakeries, breweries, barns, dairies, and greenhouses are favorite places for molds to grow. Loggers, mill workers, carpenters, furniture repairers, and upholsterers often work in moldy environments.

Which molds are allergenic?

Like pollens, mold spores are airborne allergens that are abundant, easily carried by air currents, and allergenic in their chemical makeup. Found almost everywhere, mold spores in some areas are so numerous they often outnumber the pollens in the air. Fortunately, however, only a few dozen different types are significant allergens. In general, *Alternaria* and *Cladosporium (Hormodendrum)* are the molds most commonly found both indoors and outdoors throughout the United States. *Aspergillus, Penicillium, Helminthosporium, Epicoccum, Fusarium, Mucor, Rhizopus,* and *Aureobasidium (Pullularia)* are also common.

Are there other mold-related disorders?

Fungi or microorganisms related to them may cause other health problems similar to allergic diseases. Some kinds of *Aspergillus* may cause several different illnesses, including both infections and allergy. These fungi may lodge in the airways or a distant part of the lung and grow until they form a compact sphere known as a "fungus ball." In people with lung damage or serious underlying illnesses, *Aspergillus* may grasp the opportunity to invade the lungs or the whole body. In some individuals, exposure to these fungi also can lead to asthma or to a lung disease resembling severe inflammatory asthma called allergic bronchopulmonary aspergillosis. This latter condition, which occurs only in a minority of people with asthma, is characterized by wheezing, low-grade fever, and coughing up of brown-flecked masses or mucus plugs. Skin testing, blood tests, X-rays, and examination of the sputum for fungi can help establish the diagnosis. Corticosteroid drugs are usually effective in treating this reaction; immunotherapy (allergy shots) is not helpful.

Indoor Air Regulations and Mold

Standards or Threshold Limit Values (TLVs) for airborne concentrations of mold, or mold spores, have not been set. Currently, there are no EPA regulations or standards for airborne mold contaminants.

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Multiple Chemical Sensitivity

Synthetic chemicals are all around us. They're in the products we use, in the clothes we wear, in the food we eat, in the air we breathe at work. Because chemicals are everywhere in the environment, it's not

possible to escape exposure. No wonder, then, that many people have become sensitized to the chemicals around them. For some people the sensitization is not too serious a problem. They may have what appears to be a minor allergy to one or more chemicals. Chemical sensitivity is not a true allergic reaction because IgE is not actually present. Other people are much more seriously affected. They may feel tired all the time, and suffer from mental confusion, breathing problems, sore muscles, and a weakened immune system. Such people suffer from a condition referred to as Multiple Chemical Sensitivity (MCS).

What is Multiple Chemical Sensitivity?

MCS is a disorder triggered by exposures to chemicals in the environment. Individuals with MCS can have symptoms from chemical exposures at concentrations far below the levels tolerated by most people. Symptoms occur in more than one organ system in the body, such as the nervous system and the lungs. Exposure may be from the air, from food or water, or through skin contact. The symptoms may look like an allergy because they tend to come and go with exposures, though some people's reactions may be delayed. As MCS gets worse, reactions become more severe and increasingly chronic, often affecting more bodily functions. No single widely available medical test can explain symptoms. In the early stages of MCS, repeat exposure to the substance or substances that caused the initial health effects provokes a reaction. After a time, it takes less and less exposure to this or related chemicals to cause symptoms. As the body breaks down, an ever-increasing number of chemicals, including some unrelated to the initial exposure, are found to trigger a reaction. MCS affects the overall health and feeling of well being of those with the disorder. It typically impairs many bodily functions including the nervous system and digestion. Each individual affected by MCS has a unique set of health problems. A chemically sensitive person may also have other preexisting health conditions. Many affected people experience a number of symptoms, in relation to their chemical exposures. MCS may result from a single massive exposure to one or more toxic substance or repeated exposures to low doses. People with MCS may become partially or totally disabled for several years or for life.

Treatment

MCS is difficult for physicians to define and diagnose. There is no single set of symptoms which fit together as neither a syndrome, nor a single diagnostic test for MCS. Instead, physicians should take a complete patient history that includes environmental and occupational exposures, and act as detectives in diagnosing this problematic condition. After the onset of MCS, a person's health generally continues to deteriorate. It may only begin to improve once the chemical sensitivity condition is uncovered. While a number of treatments may help improve the baseline health status for some patients, at the present time, there is no single "cure" except avoidance.

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Pollen Allergy

Pollen

Each spring, summer, and fall, tiny particles are released from trees, weeds, and grasses. These particles, known as pollen, hitch rides on currents of air. Although their mission is to fertilize parts of other plants, many never reach their targets. Instead, they enter human noses and throats, triggering a type of seasonal allergic rhinitis called pollen allergy, which many people know as hay fever or rose fever (depending on

the season in which the symptoms occur). Of all the things that can cause an allergy, pollen is one of the most widespread. People with pollen allergies often develop sensitivities to other troublemakers that are present all year, such as dust mites. Year-round airborne allergens cause perennial allergic rhinitis, as distinguished from seasonal allergic rhinitis.

What is pollen?

Plants produce microscopic round or oval pollen grains to reproduce. In some species, the plant uses the pollen from its own flowers to fertilize itself. Other types must be cross-pollinated; that is, pollen must be transferred from the flower of one plant to that of another plant of the same species. Insects do this job for certain flowering plants, while other plants rely on wind transport. The types of pollen that most commonly cause allergic reactions are produced by the plain-looking plants (trees, grasses, and weeds) that do not have showy flowers. These plants manufacture small, light, dry pollen granules that are custom-made for wind transport.

Where is pollen most common?

Most allergenic pollen comes from plants that produce it in huge quantities. A single ragweed plant can generate a million grains of pollen a day. Samples of ragweed pollen have been collected 400 miles out at sea and 2 miles high in the air. The chemical makeup of pollen is the factor that determines whether it is likely to cause hay fever. For example, pine tree pollen is produced in large amounts by a common tree, which would make it a good candidate for causing allergy. The chemical composition of pine pollen, however, appears to make it less allergenic than other basic types. Because pine pollen is heavy, it tends to fall straight down and does not scatter. Therefore, it rarely reaches human noses. Among North American plants, weeds are the most prolific producers of allergenic pollen. Ragweed is the major culprit, but others of importance are sagebrush, redroot pigweed, lamb's quarters, Russian thistle (tumbleweed), and English plantain. Grasses and trees, too, are important sources of allergenic pollens. Although more than 1,000 species of grass grow in North America, only a few produce highly allergenic pollen. These include timothy grass, Kentucky bluegrass, Johnson grass, Bermuda grass, redtop grass, orchard grass, and sweet vernal grass. Trees that produce allergenic pollen include oak, ash, elm, hickory, pecan, box elder, and mountain cedar. It is common to hear people say that they are allergic to colorful or scented flowers. In fact, only florists, gardeners, and others who have prolonged, close contact with flowers are likely to become sensitized to pollen from these plants. Most people have little contact with the large, heavy, waxy pollen grains of many flowering plants because this type of pollen is not carried by wind but by insects such as butterflies and bees.

When do plants make pollen?

One of the most obvious features of pollen allergy is its seasonal nature--people experience it symptoms only when the pollen grains to which they are allergic are in the air. Each plant has a pollinating period that is more or less the same from year to year. Exactly when a plant starts to pollinate seems to depend on the relative length of night and day--and therefore on geographical location--rather than on the weather. (On the other hand, weather conditions during pollination can affect the amount of pollen produced and distributed in a specific year.) Thus, the farther North you go, the later the pollinating period and the later the allergy season. A pollen count, which is familiar to many people from local weather reports, is a measure of how much pollen is in the air. This count represents the concentration of all the pollen (or of one particular type, like ragweed) in the air in a certain area at a specific time. It is expressed in grains of pollen per square meter of air collected over 24 hours. Pollen counts tend to be highest early in the morning on warm, dry, breezy days and lowest during chilly, wet periods. Although a pollen count is an approximate and fluctuating measure, it is useful as a general guide for when it is

advisable to stay indoors and avoid contact with the pollen.

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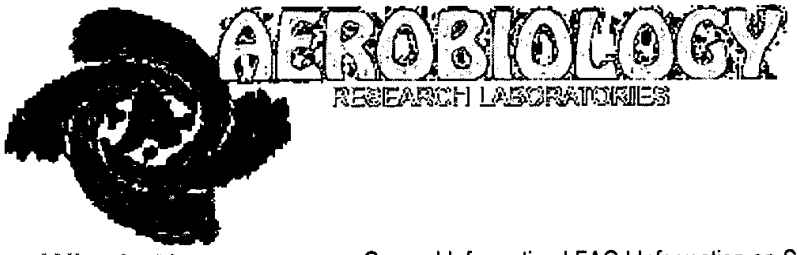
Call 1-800-771-ACHOO (2246)

Allergy Store



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General Information | FAQ | Information on Specific Pollen

The Pollen FAQ

Frequently asked questions regarding pollen and pollen allergenicity. Please choose a question from the list below, or scroll down to read our answers.

What is pollen?

Pollen grains are small and cannot be seen by the naked eye. Sometimes trees release so much pollen in the air that a cloud of pollen can actually be seen. Pollen grains are the male reproductive bodies of plants (like the sperm of animals) by which the female flowers are fertilised. This is how the plant reproduces and keeps the species alive. (top)

How is pollen collected?

There are different types of technologies or methods used to collect pollen from the air. The most commonly used is a simple technology where a small plastic rod, coated with grease, rotates in the air and the pollen sticks to it as it spins. (top)

How does one count poll n?

The rods are stained and placed on a microscope, so one can see the pollen, count and identify them. (top)

How does one obtain a count for all rg ns that ar in outd or air?

A greased rod is spun in the air at regular intervals for 24 hours. When the rod is removed the laboratory counts the number of pollen grains on the rod. A known volume of air has been sampled and the count is reported in grains per cubic meter of air. This number is used to give an indication of whether or not the concentration of certain pollen is low or high. This should help Doctors and allergy sufferers, who follow allergy reports, to know which pollen are in the air and what the concentration is. (top)

Is pollen from certain trees, flowers or weeds more allergenic than others?

The simplest answer is yes. Pollen that get airborne are of more concern than those that are insect pollinated. Airborne pollen are the ones that cause allergies because they can be inhaled. Insect pollinated ones, such as from flowers, are not of much concern. Flowers are only a problem to highly sensitised individuals who are exposed by touching the plant and perhaps inhaling lots of pollen from their hands. Often what people are reacting to when it comes to decorative flowers and plants is the scent and not the pollen. It is also important to note that pollen from certain trees and weeds and grasses are more allergenic than others. Boxelder (a species of maple) is highly allergenic, whereas the pollen from poplar is less so. Ragweed is one of the most highly allergenic plants. (top)

Do all plants that produce pollen cause allergic reactions?

No. The types of pollen that most commonly cause allergic reactions are produced by the plain-looking plants (trees, grasses and weeds). The showy plants that produce colourful flowers are insect pollinated and usually do not get airborne. Pollen from plants which are wind pollinated, however, are not all allergenic. The protein found in certain plants that are wind-pollinated are what a person's immune system is reacting to. This protein is not only found in the pollen but also in plants parts that get airborne. This is why someone who has allergies to grass reacts when they walk on a freshly cut lawn. (top)

Why are some pollen allergenic while others are nly minor allergens or do not caus allergic reacti ns at all?

The chemical makeup, or protein, of pollen is the factor that determines whether it is likely to cause allergic reactions. (top)

I have all rgi s, why are they wors som y ars than oth rs?

The seasons for trees, weeds and grasses are very different from year to year. This is largely due to the affect of weather and the environmental stresses on determining how much pollen will be produced and is released from year to year. An example is if we have a very cold wet spring when the trees are pollinating it will have a huge affect on the amount of pollen found in the air. This will usually cause a short pollen season with low pollen levels. Another example is if we have a very dry spring and summer than the amount of grass pollen in the air will be lower since the plant biologically goes dormant if it does not have enough water. If the grass is not growing it also will not have as much pollen to release. (top)

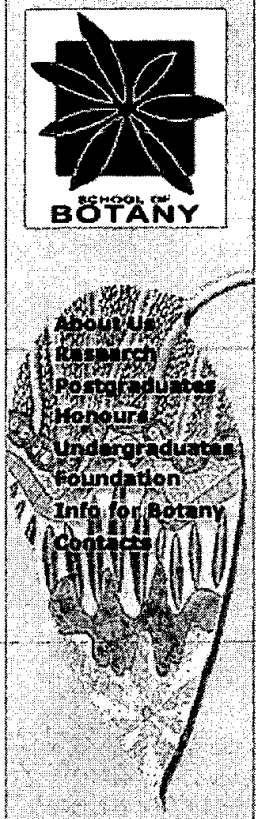
What is the significance of pollen counts?

Pollen counts are important as they give vital information as to what is in the air. Allergists and their patients can compare what is actually in the air and the allergy symptoms and evaluate the effectiveness of treatment programs. (top)

How can I avoid pollen when I have allergies?

The simplest thing is to avoid being outdoors as much as possible when the particular plant you are allergic to is flowering. Air conditioners and filters are also very helpful. Antihistamines and an effective treatment program with an allergist can help in relieving some of the symptoms. (top)

- Jump to the Spore FAQ
- Read general information on pollen and related allergies
- View specific information on various pollen



Frequently Asked Questions about Pollen

What is pollen?

Pollen contains the plant's male gametes. The closest analogous cell type in a human is male sperm.

Why is there pollen in Melbourne's air?

Plants are immobile and so cannot go searching for suitable mates the way animals do. To ensure their gametes reach receptive females, plants have developed many ways of distributing their pollen. Some species use the wind to carry pollen between plants. These plants have very simple, dry flowers that do not secrete the nectar that attracts birds and insects. Wind-borne pollen can drift considerable distances. Pine pollen, for instance, can be carried hundreds of kilometres. But, then pine pollen has wings that serve as floats! Pollen of wind-dispersed species such as rye grass, the major source of pollen in Melbourne's air in the late spring and early summer, is relatively small in size, (usually <30 micrometers), has smooth outer surface, and is relatively dry and powdery. A number of factors, such as wind speed, humidity, down gradient, etc., determine how far such pollen will travel. Other plant species use insects and birds to carry their pollen between plants. These plants produce pollen that is larger in size (>30 micrometers), has extensive surface ornamentation, contains high levels of water, and also has a sticky surface. These pollen are also heavier and sometimes occur in clumps. They have less chance of drifting any distance on the wind.

Can you recommend any medication for my allergies?

The Melbourne Pollen Count does not offer medical advice. Please consult your doctor to discuss proper treatment of your allergy symptoms.

How are pollen counts done?

We use an air-sampling device called a Burkard spore trap to capture airborne pollen on a glass slide, where it can be stained with a dye and counted using a microscope. During the season, a slide is removed from the trap at the same time each day and counted twice. The first counts all types of pollen and the second just the grass pollen (which has a distinctive shape). Our daily pollen count is a report of grass/all kinds of pollen (as

grains per cubic metre of air) caught in the trap in the previous 24 hours. Our pollen forecast is based on this count and on the weather forecast for the next 24-hr period.

Why is pollen counting only done over spring and summer?

We usually begin reporting in October, when grasses start flowering and there are measurable amounts of grass pollen in the air. By the middle of summer when the grass has died off, there are again minimal amounts of amount of grass pollen in the air. During the grass pollen season the count is done daily and the information distributed through various media outlets.

How many pollen-counting stations are there in Victoria?

The only station currently operating in Victoria is at the University of Melbourne. Other capital cities have their own pollen count stations.

What factors affect the daily pollen count?

A number of factors affect the count, including daily fluctuations in temperature, wind conditions, humidity and precipitation, and of course the biology of the plants themselves. Many plants flower in the morning so concentrations of airborne pollen are usually highest between 5 a.m. and 10 a.m.

Weather conditions also affect pollen levels. The biggest factors affecting pollen counts are wind, and humidity. Melbourne's worst pollen days are characterised by hot northerly winds that bring pollen in to the city from pastures in the surrounding countryside. When the air is humid, such as during or after it rains, pollen that is small, light and dry and easily spread by wind, becomes heavy with moisture and can't travel as far.

Is the pollen season the same from year to year?

Not exactly. The beginning and end of the grass pollen season depends on the previous year's weather (how much grass is growing) and current weather (how much grass is flowering).

Where can I find the daily pollen count and why don't you put it on this site?

The daily count and forecast are not available on our webpage because very few people even know this site exists. Instead we supply the information each day of the pollen season to key media outlets in Melbourne. This means we reach many more people than we would if we just used the web. The daily count and forecast are shown nightly in the weather section of the Channel 7 and 9 News, and appear the following day in the Herald Sun and Age.

Where can I get historical data (past pollen counts, etc.)?

Files of pollen counts for the past several seasons can be downloaded from the count data page.

[Home] [Intro] [Data] [FAQ]

THE UNIVERSITY OF MELBOURNE

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Saint Louis COUNTY HEALTH

Pollen and Mold Center

Service Connections:
Services Finder
Online Media Center
News Room

Division Connections:
Administration
Health Centers
Public Health Services
Environmental Services
Medical Examiner

Community Connections:
Health Education
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Frequently Asked Questions (FAQ's)

Return to Pollen and Mold Center

What is pollen?

Pollen is the male "seed" of a plant that appears as a dust. It can be transferr or the wind for plant reproduction.

Who can count pollen and mold?

Only certified counters can read pollen and mold. Each counter must pass certification course provided through the Harvard School of Public Health accredited by the American Academy of Allergy, Asthma and Immunology Environmental Health Laboratories has certified counters on staff. Meteorol specialists, physicians, and individuals have relied on the Saint Louis County D Health for this data since 1960. We report our data to news and weather c health organizations such as the American Lung Association.

How are pollen and mold collected?

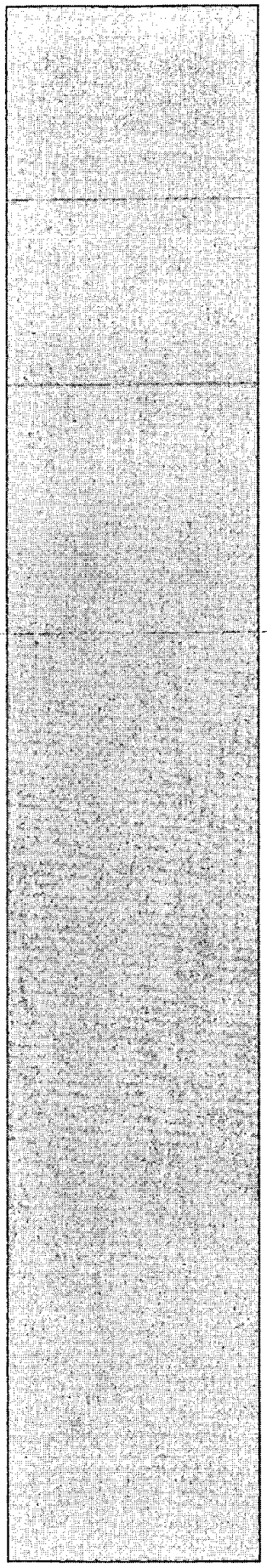
Counters use air sampling equipment to capture airborne pollen and mold. Environmental Health Laboratories switched from using a rotorod impaction d a Burkard slit-type volumetric spore trap. The rotorod sampled only at specific while the Burkard is able to continuously sample over a 24 h

How does the Burkard sampling device work?

The device is mounted on the roof of a centrally located County building a obstructions. It uses suction to pull air through a slit-type opening. Inside greased, flat surface (a collection tape) that advances in increments over time. surface collects any particles that are sucked in with the air.

How ar poll n and mold count d?

The collection tape is removed from the sampling device and brought to t Here it is stained and prepared for analysis. The sample can then be magnifie



count the pollen grains. For some mold spores, the sample must be magnifie to be seen and counted. Using the exposure time, the volume of air sam number of pollen grains or mold spores counted, calculations can be made to number of particles per cubic meter of air sampled. This is the number re laboratory.

Why is there not always a count availabl ?

There are many reasons why no count is available at various times. Some pos include technical difficulty with the sampling device, inclement weather, the unreadable, illness or absence of the laboratory's certified counter(s), or the closed for the holidays.

Why do pollen and mold counts vary so much from day to day?

Changes in temperature, wind conditions, humidity, or precipitation can affe greatly.

- Temperature: A sudden temperature drop lowers the pollen count significantly. are seasonal. Trees are dominant in the spring, grasses occur in late spring anc summer, and weeds grow from late summer until the first hard frost.
- Wind: Pollens are small, light, and dry so they are easily spread by wind. The di pollen travel can depend on whether the wind is strong or calm that day.
- Humidity: When the air is humid, pollen becomes damp and heavy with moistur still and on the ground.
- Precipitation: Rains tend to "cleanse" the air of pollen. When the pollen is wet, it heavy with moisture keeping it on the ground.

Are pollen seasons the same every year?

Generally tree, grass, and weed seasons are similar every year in the s However, the intensity can differ depending on the current weather, the pr weather, and other environmental factors. Typically, trees pollinate earliest fro May, grasses follow in May to mid-July, and weeds peak from late summe

How do the pollen counts apply to my area if I live x miles from a countin

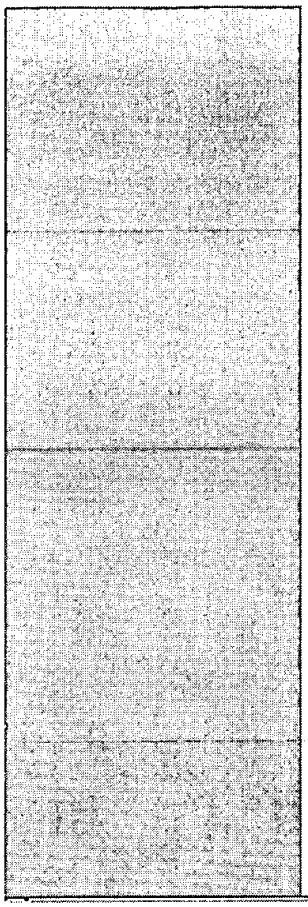
If the climate and geography are similar, the counts should be a good indi area. Keep in mind that samples taken from an urban area, where there is litt can differ from samples taken from a rural area, where there is more poll vegetation.

What is an allergy?

An allergy is an abnormal reaction to an ordinarily harmless substance calle Common allergens include pollens, molds, dust mites, animal dander, foods, cockroach droppings, and insect stings/bites. You may be allergic to one or m When an allergen is absorbed into the body of an allergic person, their bod itself of the allergen. The immune system initiates a defense which causes sy as runny nose, watery eyes, congestion, itching, and sneezing.

Will moving help my allergies?

When a person with allergies moves to another location, they will likely be exp set of allergy triggers. In some cases, the new symptoms may be more tol intense. Keep in mind that it can take months or years to become allergic to a Seasonal allergy sufferers may be able to find temporary relief by vacationi peak of pollen season to a different climate or a more pollen-free area such



bodies of water.

How can I lessen my xposure to poll n and mold?

During the peak of the pollen or mold season that affects you, try following the:

- Keep windows closed at night.D
- Minimize early morning outdoor activity when most pollen is released (between !
- Keep your car windows closed when traveling.
- Stay indoors when the pollen count is high and on windy days when dust and po scattered.
- Vacation during the peak of pollen season to an area where there is less pollen, beach.
- Take any medications your allergist recommends as prescribed.
- Do not rake leaves, mow lawns, or be around freshly cut grass. This stirs up pol
- Do not hang laundry outside to dry. Pollen and mold will collect in them.
- Keep indoor plants to a minimum and never overwater if allergic to mold. Wet s mold growth.

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Allergens: Pollen

What is pollen?

Pollen is the tiny egg-shaped male cells of flowering plants, including trees, grasses, and weeds. Pollen is microscopic in size.

Pollen is the most common cause of seasonal allergic rhinitis, sometimes known as **"hay fever."**

Which plants produce pollen that cause allergic reactions?

Plants that have powdery granules of pollen that are easily blown by the wind, such as:

- **Trees:** oak, western red cedar, elm, birch, ash, hickory, poplar, sycamore, maple, cypress, walnut, and others.
- **Grasses:** timothy, Bermuda, orchard, sweet vernal, red top, some blue grasses, and others.
- **Weeds:** ragweed, sagebrush, pigweed, tumbleweed, Russian thistle, cockleweed, and others.

Most flowering plants, such as roses, have heavier, waxy pollens that are not as easily wind-blown.

When is "pollen season?"

Each plant has a pollen season. It usually starts in the spring, but may begin as early as January in the southern areas of the US. The season usually lasts until October.

Can allergic rhinitis in poll n season b prev nted?

To lessen the effects of allergic rhinitis during pollen season, the American Academy of Allergy, Asthma and Immunology suggests the following:

- Keep windows closed at night and use air conditioning, which cleans, cools, and dries the air.
- Minimize outdoor activities early in the morning, between 5:00 and 10:00 a.m., when pollen is most prevalent.
- Keep cars windows closed when traveling.
- Take a vacation to an area [such as the ocean] where pollen is not as prevalent.
- Take the medications prescribed by your physician.
- Don't spend much time outdoors when the pollen count is high.
- Don't rake leaves during pollen season.
- Don't hang bedding or clothing outside to dry.
- Don't grow too many indoor plants.

This content was last reviewed by a University of Maryland Medicine expert on
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